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ECZEMA AND PRURITUS IN THE AGED*

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SINCE the average span of life has increased steadily and the population has come to be made up of a larger proportion of older people, medicine in general has paid more attention in the past decade or so to the problems presented by this group, and the term "geriatrics" has become commonplace in medical literature. Numerous problems peculiar to old age have been subjected to special study, and articles on a wide range of these have been published. Eczema and pruritus in the aged have not received a great deal of special attention in the literature largely because they present essentially the same problem in old people as in other age groups except for the clinical picture produced by senescent skin changes themselves.

The words, "eczema and pruritus," embrace a very wide field and in their discussion a whole treatise on general dermatology could properly be included, since a majority of dermatoses are eczematous or dermatitic and most are pruritic. This paper, however, is limited to the chief conditions producing a more or less generalized pruritus.

It might be well to make a few prefatory remarks about the mechanism of "itch." Itching is a distinctive cutaneous sensory disturbance peculiar to the integument and does not depend on the presence of cutaneous eruption for its existence. It is a complex sensation composed of a blending of touch and pain, and can be perceived only when both the touch and pain transmission tracts are functioning. It is apparently largely a thalamic rather than cortical sensory sensation and can occur in sleep and semiconscious states. Scratching during sleep in many of the pruritic dermatoses is, of course, a common observation. Perception of itch depends on an attention factor and a threshold susceptibility factor; the former controls the slight sensations present in even normal skins, and these are therefore not perceived as itch. In long-standing pruritus, the threshold is lowered, and fatigue states affect the attention factor, so that the treatment problem is not an easy one.

It should first be said that a diagnosis of senile eczema or pruritus is too often made just because the patient is aged. Instead, as in younger patients, one should attempt to differentiate and classify the aged patient's condition. Even when one is presented with the picture of pruritus without gross cutaneous change in a thin, dry, aged and atrophic skin, the diagnosis of senile pruritus should not be made too readily without a consideration and elimination of other possible explanations; the patient may have subthreshold jaundice, for example, from early obstruction by a cancer.

TYPES OF PRURITUS

Senile pruritus occurs in both men and women, usually over sixty years of age. This age is an arbitrary one, since, obviously, some patients have senescent cutaneous changes beginning at an earlier age and others have little discernible atrophy even in the sixties. The exciting cause of senile pruritus is atrophy of the entire thickness of the skin, including its appendages, which results in a thin, dry, irritable skin and leads to generalized itching.

Psychoses and psychoneuroses are primarily psychiatric rather than dermatologic problems, but the average practitioner can certainly do more even in an anxiety neurosis than dismiss the patient with the statement, "It's all in your imagination." Acarophobia (fear of scabies or imaginary infestation with mites or scabies) should be recognized because much can be accomplished by authoritative and convincing reassurance. A patient with acarophobia almost invariably picks

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from the skin pieces of lint, crusts and so forth, and is convinced that these are mites; he usually brings to the physician some of these specimens in a pillbox or carefully wrapped in paper. Even if the nature of the specimen is grossly obvious, it should be studiously examined with a lens or microscope, preferably in the presence of the patient. If the true situation is then explained and the patient reassured, this in itself effects a cure in most cases unless there is a deeper underlying "sense of guilt" or other psychiatric problem.

Diabetes can be diagnosed or ruled out by urinalysis and blood-sugar determination, and the glucose-tolerance test, if indicated. Diabetes does not produce a distinctive eruption in patients who have pruritus because of it. Carbuncles, furuncles and other pyogenic infections are likely to occur, but may be entirely absent in the patient presenting himself because of pruritus. Necrobiosis lipoidica diabeticorum is a rare dermatosis observed in diabetic or prediabetic patients, but its appearance is so distinctive that it could hardly be mistaken for eczema and need not be discussed here.

Jaundice is a well-known cause of generalized itching, and no one would fail to recognize this relation in a fully developed icterus. However, it must be remembered that pruritus can be present and troublesome when the skin has no observable yellowish tint and the scleras appear normal. An icteric index determination is helpful, and a full medical workup from the standpoint of the biliary system should be done if indicated.

The *lymphoblastoma group* includes Hodgkin's disease, lymphatic and myelogenous leukemia, lymphosarcoma and mycosis fungoides. These are fortunately rare, but those who practice dermatology have at some time seen one or another of these in a patient who was considered to have "eczema." The clinical pictures are not distinctive enough to warrant taking time to attempt to describe or differentiate them, except in the tumor stage of mycosis fungoides. But even in the early stages of mycosis fungoides, which may last for years, a definite diagnosis cannot be made on clinical appearance alone. The type of patient who comes under the scope of this discussion usually presents a fairly extensive erythrodermia or exfoliative dermatitis. The skin may show definite infiltration, or this may not be evident; there may be an accompanying lymphadenopathy, and the pruritus may be intractable. Diagnosis depends on the study of the cellular constituents of the blood and on biopsy of the skin and lymph nodes. It must be borne in mind, of course, that the skin can show typical and pathognomonic leukemic infiltration in the presence of a normal picture in the circulating blood.

Dermatitis hiemalis (winter itch) and *bath dermatitis* can, for convenience, be considered together. They occur at any age, but the senescent dry atrophic skin is especially vulnerable. Although some persons apparently react with pruritus to cold itself, most patients have their dermatitis or pruritus because of excessive defatting and drying of their skins by means of soap and water and the dry overheated air of dwellings. The trunk and the extensor surface of the extremities are especially involved. Only the evidence of scratching and a mild ichthyotic change may be present, or a patchy dry superficial erythematosquamous dermatitis, at times even eczematized. In warm weather, the condition is ameliorated or disappears because the overdried air factor is no longer operative, and perspiration partly assumes the role of lubrication carried on by sebaceous activity in younger skins.

Urticaria is distinguished by the presence of wheals, with which everyone is so familiar that the eruption cannot be confused with eczema. However, the persistence of uticaria is due to recurring crops of lesions, and the individual lesion may reach full development and undergo involution in a few hours, so that no trace of wheals may be present at the time of examination. A careful history, however, and subsequent observation establish the diagnosis and rule out senile pruritus. Giant hives or angioneurotic edema is a not infrequent accompaniment of chronic urticaria.

Drugs, such as morphine and cocaine, taken by addicts can produce generalized pruritus. History and concomitant findings point the way to the diagnosis. The various drugs used therapeutically that can produce a dermatitis medicamentosa or pruritus are too numerous to list in a paper of this scope.

Parasites that are most commonly encountered in this vicinity include scabies and pediculi. Scabies is produced by a macroscopically visible mite that burrows into the skin to deposit its ova. When a classic burrow is found, diagnosis is easy. Usually, however, scratching destroys these, and one must depend on the history and the distribution of the eruption. Itching is most intense after the patient is in bed, when the warmth of the bedclothes stimulates the mites to activity. The thin-skinned regions are characteristically involved, and the sites of predilection are as follows: finger webs, flexor surfaces of the wrists, axillary folds, waistline, buttocks, nipples (in women) and penis (in men). The face and neck are spared except in infants. The mite can be recovered from a burrow, but this is by no means as easy as most writers lead one to believe. There

are three common kinds of pediculi: *Pediculus capitis*, *P. corporis* and *P. pubis*. The first attacks only the scalp, and the diagnosis is made by the finding of the adult louse or "nits," which are sacs containing the ova and are attached to hair shafts. *P. pubis* attacks pubic hairs primarily, but is often found in the axillas, occasionally on the hairs of the chest and abdomen, and rarely in the eyebrows and lashes. The adult louse is easily seen with a lens at the base of a hair, with its head buried in the follicle. The nits are attached to hairs but are smaller than those of head lice. *P. corporis* does not live on the skin but in the patient's clothing, especially about the seams. It produces a nondescript scratch dermatitis especially about the shoulders, interscapular region, waistline and upper portions of the buttocks; diagnosis is confirmed by examination of the underclothes for the parasite. Chiggers, straw itch, tick bites and so forth are not prevalent in Massachusetts.

Psoriasis can be very pruritic when it is acutely generalizing. If small discrete lesions are present, the characteristic micaceous scale and capillary bleeding points can usually be found in well-developed lesions. When the process has turned into a universal exfoliative dermatitis, as it does only rarely, diagnosis cannot be made without the help of biopsy or previous knowledge of the pre-existing psoriatic eruption.

Lichen planus is characterized by pruritus, but diagnosis is easy even in extensive eruptions because of the presence of violaceous polygonal flat lichenoid papules with a characteristic sheen and almost no scale. When the papules are large or confluent, fine grayish lacelike streaks (known as Wickham's striae) are usually easily seen and are practically pathognomonic. In the extremely rare case in which lichen planus evolves into a universal exfoliative dermatitis, the diagnostic problem is the same as in psoriasis.

Dermatitis herpetiformis is a rare dermatosis of multiform character but showing almost always some herpetiform, grouped vesicles. It runs a chronic course, and the skin tends to become pigmented at the sites of involvement. It is often extensive but not universal, and sites of predilection are the extensor surfaces of the extremities, the midline of the back, the shoulders, and the back and sides of the neck. Diagnosis is suggested by chronicity, pigmentation and herpes-like grouped vesicles or blebs.

Contact sensitivity to wool or other articles of clothing can produce pruritus or eczematous eruptions. Careful history taking and indicated patch-test studies are essential for a positive diagnosis.

Treatment

In general, treatment should be aimed at the causative and contributing constitutional and psychoneurogenous factors in generalized itching, and antipruritics used as adjuncts.

Of the antipruritics, phenol, menthol, camphor and liquor carbonis detergens are in widest use and least likely to do harm. The cocaine derivatives include benzocaine, nupercaine, butyn, diothane hydrochloride and holocaine, all of which not uncommonly produce contact dermatitis after the patient's skin has developed a sensitivity to them, and should be avoided if possible. Witch-hazel water and alcohol have antipruritic value, and plain hot or ice water often controls a localized itching bout. The cornstarch or oatmeal bath is useful in generalized itching and should usually be followed by the application of an oily lotion.

In senile pruritus and other pruritus without dermatitis, local medication in the form of a lotion may be employed. Although many more elegant formulas can be made up, the unavailability of their newer ingredients in neighborhood pharmacies is a distinct disadvantage so that the time-honored Pusey's calamine liniment is still a useful preparation:

℞	Liquid phenol		1.0 cc.
	Oil of bergamot		1.3
	Powdered tragacanth		5.3 gm.
	Zinc oxide		
	Calamine	āā	30.0
	Olive oil, 150.0 cc., or liquid petrolatum		90.0 cc.
	Water	q. s. ad	480.0

Make an emulsion of the oil with tragacanth. Add gradually, with constant shaking, the other ingredients already mixed.

For patients with winter or bath itch, the cornstarch bath is substituted for soap bathing, and the following lotion is prescribed:

℞	Starch		8.0 gm.
	Glycerin		4.0
	Witch-hazel water		16.0 cc.
	Oil of sweet almond		60.0
	Olive oil		
	Lime water	āā q. s. ad	240.0

If the antipruritic effect needs to be enhanced, 1 to 2 per cent phenol and 0.5 or 0.25 per cent menthol or camphor may be added; or the following lotion, which contains tar, may be substituted:

℞	Phenol		2.4 cc.
	Camphor		1.2 gm.
	Glycerin		2.4 cc.
	Liquor carbonis detergens		7.2
	Starch		12.0 gm.
	Olive oil		
	Lime water	āā q. s. ad	240.0 cc.

For the dry erythematosquamous dermatitic patches in these patients, the following ointment may be prescribed:

℞ Salicylic acid		0.3 gm.
Liquor carbonis detergens		3.0 cc.
Aquaphor		
Rose water ointment	āā q. s. ad	30.0 gm.

If lichenification is present in chronically scratched areas, 5 to 10 per cent oil of cade or pine tar may be used in the above formula; or crude coal tar ointment may be prescribed as follows:

℞ Crude coal tar		2.0 cc.
Zinc oxide		2.0 gm.
Mix and let stand 24 hours		
Starch		8.0
Yellow petrolatum	q. s. ad	30.0
Make an ointment		

If the patches are frankly eczematized, the following formula is useful:

℞ Ichthyol		0.9 gm.
Zinc oxide ointment		30.0

For scabies, an ointment containing sulfur and balsam of Peru is efficacious:

℞ Precipitated sulfur		12.0 gm.
Balsam of Peru		12.0 cc.
Lanolin		30.0 gm.
Benzoinated lard	q. s. ad	120.0

For children, betanaphthol may be substituted for the sulfur in a strength of 3 to 5 per cent, an equal concentration of Peru balsam being employed. Successful cure depends on careful instruction of the patient. A hot soap bath should be taken the first night, and all crusts and vesicle tops scrubbed off; the ointment should then be rubbed into the entire skin surface from the neck down. On the next morning and night, the ointment is reapplied, and on the following morning, a hot soap bath is taken and all underclothing and bed linens cared for by boiling or dry cleaning. One of the above oily lotions may be used if there is irritation from the treatment course. One course rarely fails to cure and, in any event, should not be repeated in less than ten days.

P. capitis is best treated by the time-honored turbanning of the scalp with equal parts of olive oil and kerosene overnight, followed by shampoo and removal of the nits by soaking with vinegar and the use of a fine-tooth comb. *P. corporis* requires attention to the clothing and one of the above lotions for the scratch dermatitis. *P. pubis* should not be treated with blue ointment because of the intense irritation provoked, but mercury may be used as a 1:500 solution of the bichloride and the nits soaked off with vinegar.

Generalizing psoriasis must be treated with caution, and no vigorous antipsoriatic medications should be employed. Autohemotherapy is useful at intervals of three to seven days; the starch bath may be used and followed in the guttate variety by an ointment containing 2 per cent each of salicylic acid and ammoniated mercury in a cold cream or Aquaphor base.

In lichen planus, irradiation is of distinct help but should be employed by an expert only. Mercury or arsenic by mouth or injection is indicated. A combination of the two metals in the form of mercury salicylarsenate (Enesol) is widely used intramuscularly, but occasional reactions occur. Local applications are of little avail and are indicated only to control pruritus.

Dermatitis herpetiformis is notoriously resistant to treatment, although some promising results have been obtained with the sulfonamides. Sulfur baths (a 2.5-cm. cube of commercial sulfurated potash being added to the tub), antipruritic lotions and arsenious acid by mouth in the form of Asiatic pills often produce a remission.

Summary

Eczema and pruritus in the aged present the same problems as in other age groups, in addition to the factor of dry atrophic skin with increased vulnerability.

Generalized pruritus is produced by numerous conditions, and the diagnosis of "senile pruritus" should be made only after other causes have been searched for and excluded.

Therapy should not only consist of local applications but also aim at the underlying factors.

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INTRACRANIAL ANEURYSM OF THE INTERNAL CAROTID ARTERY*

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RECENTLY, a patient suffering from an intracranial aneurysm of the internal carotid artery entered the Peter Bent Brigham Hospital. So much interest was aroused from the study of this patient that I was stimulated to search out and review all similar cases admitted to the hospital, as well as those reported by others, in an effort to arrive at the proper management of this unusual condition. This paper is presented to summarize the findings and to report in detail on the most recent case.

Case Report

A 51-year-old married woman entered the Surgical Service on June 9, 1941, complaining of a "buzzing noise" over the left side of her head of 4 months' duration. The past history was essentially irrelevant. The patient had always enjoyed good health. There was no history of syphilis.

Four months before entry, the patient awoke in the morning with a severe headache. She had had numerous headaches for many years and did not consider it unusual. That night, when she bent over to untie her shoelaces, she suddenly had the sensation of a "light bulb bursting" in her head. This sensation lasted only a few seconds, but immediately afterward, for the first time, she was conscious of a rhythmic "buzzing noise" over the entire left side of her head. This noise was present constantly thereafter and often kept her awake at night. Four weeks before admission, she noticed that she had double vision, and 3 weeks later, she had a single momentary sensation, like an electric shock, in her right arm. There were no convulsions, hemiparesis or bouts of unconsciousness at any time.

Physical examination on admission showed an intelligent, well-developed and well-nourished woman in no distress. There was a loud bruit, systolic in time, over the left side of the head anteriorly and over the left common carotid artery. A systolic thrill was palpable over the left common carotid artery. The lateral rectus muscle of the left eye was paralyzed, but the other extraocular muscles were normal. The pupils were equal and reacted normally to light and accommodation. Ophthalmoscopic examination showed normal eye grounds, and the visual fields were normal. The blood pressure was 130/90. Abdominal and pelvic examinations were negative. Complete neurologic examination was negative except for paralysis of the 6th cranial nerve on the left.

Examination of the blood showed a red-cell count of 4,800,000 with a hemoglobin of 80 per cent (Sahli), and a white-cell count of 6200. The blood volume was normal. Lumbar puncture showed normal pressure and normal dynamics. The cerebrospinal fluid was water-clear and showed no increase in white cells and no red blood cells. The spinal fluid protein was 35 mg. per 100 cc. Wassermann and Hinton tests on the blood and spinal fluid were negative. X-ray films of the skull, chest and heart showed no abnormalities.

The impression on admission was that the patient had an intracranial aneurysm of the left internal carotid artery.

While the patient was in the hospital, the left common carotid artery was compressed for 20-minute periods on two occasions, without noticeable mental or physical changes. It was then believed that she could stand ligation of the left internal carotid artery.

Under local anesthesia, the left internal carotid artery was exposed. Diodrast was injected, and films of the skull were then made. Following this, the artery was divided, and the ends were ligated with silk. The murmur and thrill immediately disappeared, both subjectively and objectively. The report of the x-ray films of the skull was as follows:

> Films of the skull in the operating room after injection of 70 per cent Diodrast show the left internal carotid artery well outlined, appearing normal up to the area beside the sella, where there appears to be vascular dilatation 1.5 cm. in diameter. The upper edge of this, however, is not raised above the sella as is usual in aneurysms. The internal carotid artery beyond this point is not visualized.

After operation, the patient made an uneventful recovery. A minimum diplopia continued, as did the left 6th-nerve palsy, but the bruit remained absent. At follow-up 7 months later, the bruit was still absent, and there had been a constant decrease in the patient's diplopia until it occurred only when she turned sharply to the left. The patient had learned to avoid this maneuver and therefore was not troubled by diplopia. The vision in the left eye was equal to that of the right, but there still remained weakness in the external rectus muscle on that side, presumably evidence of continued slight impairment of the 6th-nerve function.

Previous Cases

This review of cases includes only the aneurysms located at the junction of the internal carotid artery, either at the circle of Willis or in close proximity to that region. Forty such patients, including the one reported above, have entered this hospital in the last twenty-eight years. Twenty-one cases were verified at operation or autopsy, and 19 were diagnosed clinically. One case, in addition, was verified by Diodrast injection of the internal carotid artery. Over three fourths of the cases occurred in females: there were 31 females and 9 males in the group. The cases showed a marked peak in the sixth decade of life (Fig. 1). There were twenty-seven aneurysms on the right side and thirteen on the left.

The symptoms of 36 patients, exclusive of the 4 whose aneurysms were found incidentally at autopsy, were abrupt in onset in 19 cases and in-

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sidious in 17. Headache and eye complaints were always the leading symptoms.

The third cranial nerve was involved in 23 cases (64 per cent): alone in 5 cases; with the fourth and sixth nerves in 9 cases; with the fifth nerve in 4 cases; with the fourth nerve in 2 cases; with the sixth nerve in 1 case; and with the fourth, fifth and sixth nerves in 2 cases. The sixth nerve only was paralyzed in 2 cases, the fourth and fifth in 1, and the fourth, fifth and sixth in 1.

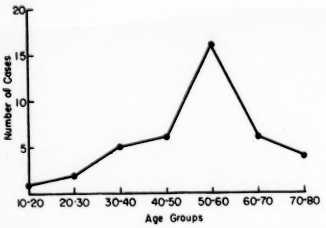

FIGURE 1. *Incidence of Aneurysms at the Junction of the Internal Carotid Artery, according to Age Groups.*

Although these 27 patients presented ophthalmoplegia of varying degrees, of the 9 cases remaining, 3 were totally blind, 3 had homonymous hemianopsia, and 3 entered in coma. Nineteen patients had hypertension, and 17 had normal blood pressure. The blood Wassermann reaction was negative in all. The absence of syphilis in patients with intracranial aneurysm, in contrast to patients with aneurysms elsewhere, has been pointed out by others.[1-3]

The presence of a bruit in the head was carefully searched for, both subjectively and objectively. Only 3 patients noticed a "buzzing noise" in the head, and in only 1 was it the chief complaint. In addition to these, a bruit over the skull was discovered by auscultation in 3 cases, so that in 6 (15 per cent) of the cases this sign was present.

The diagnosis on admission in 28 cases was intracranial aneurysm; in 5 cases, pituitary adenoma; and in 3 cases, meningioma. The neighboring symptoms, sometimes caused by a large intracranial aneurysm, may easily simulate a brain tumor, as pointed out by Symonds.[4] In 1 case, not included in this series, a diagnosis of intracranial aneurysm was made, but at operation elsewhere the source of the trouble was found to be a chromophobic adenoma of the pituitary gland.

Only 3 patients were subjected to surgical therapy. In each case, the internal carotid artery was ligated in the neck. In 1 case, the aneurysm was also opened, and muscle was packed into the sac; this patient died six months later, and autopsy showed thrombosis of the aneurysm. Of the other 2, one patient was alive and asymptomatic at the end of five years, but has not been traceable since; the other is alive seven months after operation and free of the bruit, although she still has a residual sixth-nerve palsy.

In the untreated group, 9 patients (23 per cent) died of rupture of the aneurysm, 14 died without the exact cause being known, and 14 are unaccounted for, since they did not respond to follow-up inquiries.

Discussion

Intracranial aneurysms have been the subject of considerable interest in the literature of the past few years. Some of the cases in this series have been reported by Symonds,[4] Doubler and Marlow,[5] Viets[6] and Sosman and Vogt,[7] because of interest in clinical manifestations and x-ray findings. Not until recently has an accurate clinical diagnosis of intracranial aneurysm been made. The gradual accumulation of knowledge concerning the disease and roentgenologic studies[7, 8] of the appearance of intracranial aneurysms, including cerebral arteriography introduced by Moniz,[9] have made this possible.

The syndrome of headache, either acute or recurrent, associated with paralysis of the third, fourth, fifth and sixth nerves, either individually or in combination, is strongly presumptive evidence of intracranial aneurysm of the internal carotid artery. The bruit must be searched for and, if present, is pathognomonic of the condition. Plain x-ray films of the skull may show calcification in the wall of the aneurysm. These areas of calcification are usually fine curvilinear shadows. In addition, there may be erosion of the bone adjacent to the aneurysmal sac.[7] So far as I know, there is no danger in injecting Diodrast into the internal carotid artery to visualize the aneurysm, and the vessel can be ligated at the same time.

Rupture of the aneurysm, in some cases, may be the first manifestation of the difficulty: there is sudden, severe pain along the distribution of the trigeminal nerve as the blood is spilled over the gasserian ganglion, and coma usually follows. On examination, the neck is stiff, and Kernig's sign is positive. If lumbar puncture is done, the spinal fluid is found to contain fresh red blood cells. Death usually follows rupture, although Fearnsides[1] reported that 41 per cent of his patients had one or more bouts of leakage from the aneurysm, without death.

The treatment of intracranial aneurysms has been directed toward the induction of thrombosis of the aneurysm. This has been attempted by

direct attack on the aneurysm, by ligation of the cervical portion of the internal carotid artery and by ligation by clip of the vessel distal to the aneurysm intracranially, in addition to ligation of the internal carotid artery in the neck. The direct attack on the aneurysm is usually impossible and is most dangerous.

Ligation of the cervical portion of the internal carotid artery for aneurysm was first practiced by Sir Astley Cooper in 1805. It is not a hazardous procedure in selected cases, and following it, a number of patients with intracranial aneurysm have had symptomatic relief. Schorstein,[10] who collected 60 cases of intracranial aneurysms for which the internal carotid artery had been ligated, found that in infraclinoid aneurysms ligation was a benign procedure unattended by neurologic complications or death. However, in supraclinoid aneurysms, there was a 13 per cent mortality. This mortality he ascribed to the fact that there had been recent leakage from the aneurysm or compression of the basal arteries.

Before the internal carotid artery is ligated, it should be totally occluded by pressure for ten minutes, as recommended by Matas[11] and by Dandy,[12] to determine whether or not there is any untoward effect on the cerebral circulation. Occasionally, even after the occlusion test, a late effect may follow ligation of the artery. The preoperative condition of the cerebral circulation is all important in the decision of whether the patient will tolerate ligation of one internal carotid artery.

Dandy, in 1937, first trapped an intracranial aneurysm between a silver clip distal to it and a ligature around the cervical portion of the internal carotid artery. Since then, a few similar successful cases have been reported.[12–14] It is a difficult procedure at best and not always possible; nevertheless, it is the rational method of approaching the disease.

Summary and Conclusions

A case of intracranial aneurysm of the internal carotid artery, associated with a bruit over the head and a sixth-nerve palsy, is reported. Treatment consisted in ligation of the cervical portion of the internal carotid artery.

The cases of 40 patients with aneurysm of the intracranial portion of the internal carotid artery who entered the Peter Bent Brigham Hospital in the last twenty-eight years were reviewed.

Headache and palsy of the third, fourth, fifth and sixth nerves were the leading clinical signs and symptoms. A bruit over the head was present in 15 per cent of the cases.

Ligation of the cervical portion of the internal carotid artery is advocated, provided that the cerebral circulation is sufficient after occlusion of the artery for ten minutes.

Trapping the aneurysm between an intracranial clip and a ligature around the cervical portion of the intracranial aneurysm may be attempted, but is not always possible to carry out.

Cerebral arteriography is a safe and reliable procedure to verify the presence of the aneurysm.

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GASTROINTESTINAL SYMPTOMS AND INGUINAL HERNIA*

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BEFORE operation is advised for an inguinal hernia, it is an established practice to make certain that the patient does not also present evidence of chronic pulmonary disease or partial urinary obstruction. Little significance, however, has been attached to gastrointestinal symptoms. Such symptoms occurring in patients with inguinal hernia should receive careful study. There is a tendency to attribute these complaints to the presence of the hernia and to advise repair without further investigation. This, we believe, is a dangerous policy, for not infrequently some other process, such as ulcer or cancer of the gastrointestinal tract or gall-bladder disease, is masked by the more obvious complaint of hernia.

Much of the literature suggests that gastrointestinal symptoms, such as epigastric distress, abdominal cramps, flatulence, nausea and vomiting, may be the result of an inguinal hernia that has never been incarcerated or strangulated. An uncomplicated inguinal hernia is said to produce distress varying from uneasiness in the epigastrium to actual abdominal pain in the region of the umbilicus. Nausea has been alleged to result from traction on the mesentery, and reflex disturbances of digestion — cramps, intestinal pain and gas — are described.[1-4]

On the other hand, there are those, like Behan,[5] who believe that pain, usually of a dragging nature, is of minor importance as a symptom of uncomplicated inguinal hernia. Andrews and Bissell[6] state that in at least 90 per cent of all cases of hernia pain is totally absent. In their opinion, the relief of chronic indigestion supposed to be caused by hernia has proved to be very disappointing, for it generally persists after the hernia has been surgically cured.

There are thus two opposing schools of thought: one believing that gastrointestinal symptoms can be the result of simple uncomplicated inguinal hernia; and the other that gastrointestinal symptoms are not to be explained by such an obvious reason.

Within recent months, our attention has been arrested by 2 cases of gastrointestinal cancer, overlooked at the time of herniorrhaphy. This led us to review 200 consecutive cases of uncomplicated inguinal hernia coming to operation. In studying these cases, we found that in 81, or 40 per cent, there were no complaints other than the presence of a hernia; 82 patients, or 41 per cent, had groin pain, and 37, or 18 per cent, had definite gastrointestinal symptoms that required investigation (Table 1). Thus, approximately one fifth of the patients had symptoms referable to the gastrointestinal tract, and it is with this group that we are primarily concerned.

TABLE 1. *Gastrointestinal Symptoms in Cases of Operable Inguinal Hernia.*

GASTROINTESTINAL SYMPTOMS		NO. OF CASES		PERCENTAGE OF TOTAL
Not present		81		40
Present but mild (groin pain only)		82		41
Present		37		18
Apparent cause	22		11	
No apparent cause	15		7	
No cancer postoperatively	13	6		
Cancer postoperatively	2	1		
Total		200		

Table 1 shows that 22, or 59 per cent, of the 37 patients with gastrointestinal symptoms had a plausible explanation for their complaints of abdominal pain, gas, nausea or vomiting. These were the following: momentary attacks of recognized incarceration, whose reduction relieved all symptoms; epigastric distress treated successfully by diet and alkalies that were prescribed by the family physician; and gall-bladder disease and peptic ulcer, proved by x-ray examination.

Fifteen, or 41 per cent, of these patients presented vague but persistent gastrointestinal symptoms for which no cause was found prior to operation. There was no history of incarceration; diet and medical treatment brought no relief; and x-ray examination was negative in the few cases in which it was employed. Although it is true that 11 patients in this group were relieved of symptoms following herniorrhaphy, 2 of the 15 demonstrated gastrointestinal cancer as the true basis for their complaints. If the error of first performing herniorrhaphy on a patient who also suffers from carcinoma is to be avoided, all unexplained gastrointestinal symptoms must have a thorough appraisal before operation. A review of the following cases clearly shows the need for intensive investigation of gastrointestinal symptoms in the presence of inguinal hernia.

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CASE 1. A 48-year-old man was admitted to the hospital complaining of a right inguinal hernia of 6 weeks' duration. He had experienced occasional dragging pain in the right groin, gas and sour eructations. Physical examination was not remarkable except for a right indirect inguinal hernia. Herniorrhaphy was performed, and convalescence was normal. After discharge from the hospital, gaseous eructations and epigastric distress continued. The patient was studied in the Out-Door Department, and the stools were found to be strongly positive for occult blood. X-ray studies suggested carcinoma of the stomach. The patient was therefore readmitted, 5 months after herniorrhaphy, for gastric resection for carcinoma. During the 2-month interval since gastric resection, he has had no gastrointestinal symptoms.

CASE 2. A 58-year-old man was admitted to the hospital complaining of bilateral inguinal hernias of 3 years' duration. These had been increasing in size prior to admission and were especially prominent when the patient strained at stool. The patient complained of chronic constipation, and had recently been taking increasing daily doses of laxatives. There were no groin symptoms, abdominal pain or weight loss. Physical examination showed bilateral inguinal hernias, for which bilateral herniorrhaphy was done. At the time of operation, a hard nodule was noted in the right hernial sac, which was removed for microscopic study. A metastatic carcinoma was reported, which was thought to have arisen from the gastrointestinal tract. Following recovery from herniorrhaphy, a barium enema demonstrated an obstructive lesion of the sigmoid. Subsequently, at an abdominal exploration, carcinoma of the sigmoid, with diffuse peritoneal metastases, was discovered.

These 2 cases are but examples of the pitfalls hidden by apparently simple cases of inguinal hernia. Behind the mildest symptoms may lurk serious disorders detrimental to the patient's health. This is especially true in the middle-aged and the elderly, since advancing years bring on an increased incidence of serious constitutional disease. Both urinary obstruction and chronic cough are well-recognized contraindications to herniorrhaphy, and efforts are made to remedy or control these conditions previous to operation. The physician and patient, however, are only too willing to assume that the presenting inguinal hernia is the sole cause for the patient's indigestion and to proceed with herniorrhaphy without further investigation. We believe that ulcer in the younger age groups and gall-bladder disease and cancer in the middle-aged or the elderly must be carefully excluded in patients with inguinal hernia with gastrointestinal symptoms before the hernia is repaired.

Obviously, it is impractical and financially impossible to subject every patient with an inguinal hernia and gastrointestinal symptoms to complete roentgenologic examination, however desirable this may be. But there are simple methods that will narrow down the field until only a few remain who demand further investigation. A painstaking history with special reference to the nature of the gastrointestinal symptoms and their duration often indicates patients in whom ulcer, gall-bladder disease or cancer is to be suspected. A thorough general physical examination is, of course, essential. Abdominal masses and tenderness should be sought. Rectal examination must be most thorough, and should include not only a careful evaluation of the size, shape and consistence of the prostate but also a search for masses suggestive of carcinoma in or about the rectum. Laboratory studies, including those of the blood, stool and urine, are indispensable. The finding of occult blood in a stool specimen, as illustrated in Case 1, may be the clue that abnormalities other than the hernia are present. If such measures fail completely to explain persistent gastrointestinal complaints, then the physician must carry out further investigation by x-ray.

CONCLUSIONS

Gastrointestinal symptoms occur in approximately one fifth of the patients with inguinal hernia. These symptoms, if well established, may indicate basic or antecedent causes other than hernia. The cause for such symptoms must be completely explained before operation for inguinal hernia is advised.

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EMERGENCY MEDICAL SERVICE FOR A SMALL CITY

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WATERVILLE, MAINE

IN the setting up of an emergency medical service for civilian defense, it seems decidedly advantageous to take into consideration the medical facilities and personnel that might be available. Frequently, the small community is confused, and perhaps overwhelmed, by stereotyped plans laid down by state and national authorities

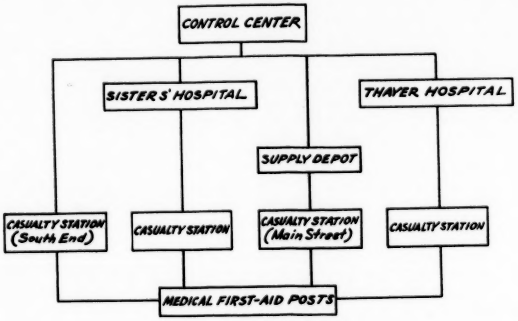

FIGURE 1.

—plans that might be possible of execution in a large city but are quite impractical for the smaller place. When one remembers the constantly diminishing supply of physicians, as more of the younger doctors are called into service, it becomes increasingly imperative that any emergency medical service be adapted to local conditions.

It was my lot to be drafted to reorganize such an emergency medical service in Waterville, Maine, a small city. The previous attempts to pattern the service on stereotyped plans had not been productive of satisfactory results.

The first step in the new plan was the utilization of the two hospitals in the city not only as casualty receiving hospitals but also as casualty stations and to furnish pools of physicians and nurses. In addition, two other casualty stations were set up to take care of sections not readily covered by the hospitals. A supply depot was established at the most centrally located casualty station.

Each casualty station, including the hospitals, is to be prepared to send out medical squads consisting of 1 physician, 1 or more nurses and 1 or more first-aid squads, and to establish first-aid posts near the scene of any incident. Stations are also prepared to handle minor cases not requiring hospitalization.

Each hospital was asked to appoint an executive medical officer, to be in charge of medical personnel, and an admitting officer, to be in charge of patients admitted because of an emergency. Provision was made for alternates. It was stated that these should be physicians of experience and executive ability, able to size up situations and make decisions quickly.

The executive medical officer specifies the duties of the physicians at his station, such as dressing room, shock room, fractures, burns and major surgery; naturally, these assignments will vary according to the type of admissions and the personnel available. He assigns physicians to be in charge of first-aid posts, when requests for such posts are received from the control center. He should exercise due judgment regarding the size and make-up of each squad, depending both on the information received of the extent of the incident and on his available supply of physicians. The executive medical officer should make a list of all physicians, nurses, first-aiders and so forth reporting at his station. This should be available for the medical service when the mobilization is over.

The admitting officer should be a physician of keen clinical judgment. He will examine cases on admission and assign them to the proper department.

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Each hospital will be notified by the control center on the yellow signal and should then proceed to ask certain of its medical personnel to assemble before the red signal. On the blue signal, the control center will ask each hospital the number of available beds. On this signal, all physicians, nurses, first-aiders and ambulances are requested to report to their stations. On the red signal, stations will telephone the control center, reporting the number of physicians, nurses and first-aiders at their posts.

The chief medical officer, with an assistant to act as clerk, handles the service from the control center. Two other physicians are familiar with this phase of the work and will be able to take charge in his absence. In fact, all key positions in the service are covered "three deep."

Three physicians are assigned to each of the casualty stations. The staffs of the two hospitals cover their respective institutions, furnishing personnel for the casualty stations, first-aid posts and the hospital itself. Graduate nurses and first-aid squads are assigned to each of the four stations. Three ambulances are assigned to each hospital, and two to each of the other stations. One or more dentists are assigned to each of the hospitals and stations.

Since, in the event of an emergency, a great deal of difficulty and confusion may be caused by distracted and hysterical relatives and friends, members of the clergy — one Catholic priest and one Protestant clergyman — have been assigned to each of the stations. An emergency morgue, in charge of an official of the Police Department, and with a Catholic priest and Protestant clergyman assigned to it, has been set up in the Municipal Armory. The supply depot, in which the reserve medical and surgical supplies, reserve emergency chests and so forth are kept, is in charge of three lay persons who are experienced in this work and who are responsible for the medical equipment of the ambulances. A pool of extra ambulances is assigned to this supply depot. The supply depot has also been charged with the responsibility of maintaining an adequate supply of various drugs, such as tetanus antitoxin and so forth, at the casualty stations. Plasma in adequate amounts is kept in storage at the two hospitals.

Telephone service must be maintained at an efficient level. When an incident requiring medical service is reported to the control center, the chief medical officer will notify the most available casualty station. The telephone operator takes the message in writing, repeats the message to the center for check, and immediately transmits it to the executive medical officer, who then dispatches one or more medical squads to the scene. As soon as this is done, the telephone operator notifies the control center as follows: "One (two) squad(s) to ——— St. at — p.m."

On arrival of the squad at the incident, a first-aid post will be set up in the most convenient location, the main essentials being light, water and communications (telephone). The squad will call its own station (hospital) if additional personnel, supplies and so forth are required and, if possible, will notify the hospital if cases are to be sent in. The executive medical officer at the station will notify the control center if an additional squad is dispatched.

If any station (hospital) is unable to furnish needed personnel or supplies, or is being depleted by the sending out of squads, the executive officer should notify the chief medical officer at the control center, who will send personnel, supplies and so forth from other stations or the supply depot.

Tags will be filled out as much as is practical at the first-aid posts. Naturally, there should not be undue delay in sending very seriously injured persons to the hospital, and many data can be obtained later at the hospital. All emergency treatment given, such as the injection of morphine or the application of a tourniquet, should be noted by marking on the forehead. Urgent cases should be marked "U." Hospitals should keep records of all cases admitted in the casualty record books furnished for this purpose.

All personnel should remain at the stations until "cleared" by the chief medical officer at the control center.

Three mobilizations, with simulated incidents, are conducted each month, in addition to a series of meetings for the entire medical service at which pertinent problems, such as "first-aid posts," "casualty stations," and "treatment of gas casualties," are discussed. It has become obvious that the main problem is one of co-ordination between the physician, the nurse and the first-aid squad. The average physician knows little about setting up or handling a first-aid post. He can take care of one casualty, but is quite at a loss when confronted with ten or twelve. Yet he is in charge and should be familiar with these problems. It is only by practice, constructively criticized, that this matter can be worked out efficiently. A system of observers — physicians competent in handling these situations — has been employed. These men not only criticize but also give instruction to the doctor, the nurse and the first-aid squad.

The first-aid organization is under the direction of a corps director and consists of five squads, one

assigned to each station and one held in reserve. Each squad is commanded by a squad leader, who is a qualified instructor. Each squad consists of four units of four men under a unit leader, who holds a Red Cross advanced certificate. One unit of each squad functions at the station, making tag records, assisting nurses, preparing dressings and splints and taking charge of patients' clothes and personal effects. Two units are allocated to ambulance duty, and one unit is held in reserve. Definite duties are assigned to each unit member, such as responsibility for ambulance equipment and splints, and the members are trained to act as a unit in handling casualties. These squads are all under the command of the corps director, who is responsible for this training. We have been fortunate in having as a director a man who has had a long experience as an instructor in first aid at the mill of a paper company that has long been recognized for its efficiency in first-aid and safety work. The director has been able to bring with him as squad leaders men who have served under him as instructors. One such squad of trained first-aiders, working under a capable leader, is worth more than a large number of well-meaning but inexperienced amateurs.

So far this plan seems to be working out very well. It is not suggested as anything new or ideal, but as a means by which a small city with limited medical personnel can meet the emergency.

Professional Building

IS TUBERCULOSIS INCREASING?*

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FOR several years, the opinion has been prevalent that tuberculosis is decreasing and that the fight against this disease has finally made definite headway. To those who are working in the field, however, there seems to be another view. At the Boston Sanatorium, the largest hospital devoted to the care of the tuberculous patient in Massachusetts, we have had an experience, over the past five years, that leads us to think that other factors have not been considered.

The total number of patients treated in 1936 was 1079; in 1937, 1088; in 1938, 1082; in 1939, 1042; in 1940, 1070; and in 1941, 1110. These patients came from two sources, the Boston Health Department and the Boston City Hospital. According to these figures, there has been a gradual increase in the admissions to the Sanatorium since 1939. The number of minimal cases consistently remained approximately 10 per cent up to 1939, when it decreased to 8 per cent; in 1940, it was 8 per cent and in 1941 7 per cent. Hence, as the total number of treated cases increased, the number of minimal cases decreased and the number of moderately advanced cases increased. This indicates either that the cases are not being discovered early enough or that the patients are not being sent for treatment until they are well along in the disease. It is pertinent to say that the minimal cases are usually discovered by extensive studies on large groups or by the study of contacts of known cases, whereas the great majority of the moderately advanced cases are picked up as the result of hospitalization for other conditions, or as the result of neglect on the part of the patient to consult his physician in time. This is equally true of the far-advanced cases.

In 1941, 29 per cent of the patients with tuberculosis who died in Boston were not reported to the Health Department until after death, and 13 per cent were reported within a month before death; the disease should have been discovered early enough to ensure information to the Health Department. In short, in 13 per cent of the known deaths from tuberculosis, the patients had no health supervision, no checkup of contacts and no restriction of activity until a month before death, and 29 per cent had no supervision whatsoever. From what is known about this disease, these patients were potentially disseminators of the tubercle bacilli for a number of months before death. When this situation is appreciated, it is not difficult to understand why 55 to 60 per cent of the deaths in 1941 at the Boston Sanatorium occurred before the patients had been at the hospital six months; their cases were so far advanced that no treatment could avail. This was not unique for 1941; it has been the same story during the last five years.

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†Clinical professor of medicine, Boston University School of Medicine; director of Fifth and Sixth Medical Services, Boston City Hospital; chief of staff, Boston Sanatorium.

This is not an isolated example of the belated discovery of tuberculosis. The May 16, 1942, issue of the *Journal of the American Medical Association* reports that of 594 men rejected for tuberculosis by the selective-service boards of Illinois, only 21 had previous records reported to the State Board of Health. At the Boston Sanatorium, 15 patients have been referred after having been rejected by their local selective-service or induction boards, few of whom suspected that they were infected. All this leads one to believe that tuberculosis will never decrease until the carriers are discovered and regulated. This will be accomplished for the male population, in a large measure, by the examinations preceding induction. This still leaves the female population to be studied; a number of these will fall into the contacts of diseased males and will be picked up in this way.

The main effort must be made with younger age groups, in the junior high schools and the secondary schools. This group should be tuberculin tested, and the reactors examined by x-ray and reported to the health authorities for follow-up. It is a simple matter for the family physician to apply a patch test, read it, and have a reactor x-rayed, but most men are reluctant to report the minimal case.

Hence, such patients who respond readily to a sanatorium regime are allowed to remain at home, where they are inadequately treated and soon become moderately or far advanced. When the problem becomes acute, they are sent to a sanatorium.

If tuberculosis is to be successfully combated, children must be patch tested, those who react must be x-rayed, and *all* cases of tuberculous infection must be reported.

464 Commonwealth Avenue

MEDICAL PROGRESS

THE BILE PIGMENTS*

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MINNEAPOLIS

THE problem of the bile pigments has always been intriguing to the clinician as well as to the physiologist, pathologist and chemist. It is quite impossible to consider this problem adequately from any one of these viewpoints. The present communication, therefore, will strive to present a survey of recent general progress in this field, with particular emphasis, however, on clinical applications, and with some attempt to correlate or compare the results of recent studies with those of the older literature, at least sufficiently for purposes of orientation. The allotted space does not permit any comprehensive review of the literature, or even an inclusion of all the various aspects of the problem. The topic of the bile pigments in relation to hemoglobin metabolism must necessarily be omitted in large part, and greater emphasis is placed on the relation of the bile pigments to jaundice and liver disease.

Reprints of articles in this series are not available for distribution, but the articles will be published in book form. The current volume is *Medical Progress: Annual, Vol. III, 1942* (Springfield, Illinois: Charles C Thomas Company, 1942. $5.00).

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BILIRUBIN

For many years, it was generally held that the transition of hemoglobin to bilirubin was characterized by a primary splitting off of the protein globin, with resulting formation of hematin. The next step was thought to be the removal of iron, followed by an opening of the porphyrin ring to yield bilirubin. This belief was founded to some extent on the ease with which hematin is obtained in vitro from hemoglobin, as well as on the known occurrence of hematin, under certain circumstances, in vivo; there was also the old idea that the iron-free hematoporphyrin of Hoppe-Seyler was an isomer and probable precursor of bilirubin. The latter investigations of the Hans Fischer school revealed clearly that the underlying porphyrin in the hemoglobin molecule, and the one standing in closest relation to bilirubin, is not hematoporphyrin but protoporphyrin. All these earlier beliefs have been dealt with in a previous review,[1] and need not be considered in more detail. Within the past few years, new concepts of bile-pigment formation have been introduced

that are based on the much firmer ground of an in vitro method of conversion of hemoglobin to bilirubin without the aid of living cells. Although it remains to be proved that this method obtains in vivo, there can be no doubt that it is sufficiently simple and physiologic in type to be quite compatible with an in vivo mechanism.

Perhaps the first observation bearing on this new concept was that of Fischer and Lindner[2] in 1926. They observed that green compounds, assumed to be similar to bile pigments, were formed when yeast and hemoglobin were incubated together. Warburg and Negelein[3] observed that when hemin is treated with pyridine-O_2-hydrazin hydrochloride a "green hemin" results. This was studied further by Lemberg et al.[4] who identified it as a biliverdin-iron-globin compound. By means of a coupled oxidation of hemoglobin or hemin with ascorbic acid, Lemberg and his co-workers[5-9] obtained bile-pigment-iron-protein compounds designated as "choleglobin," "verdohematin" or "verdohemochromogen." The first crystalline bilirubin was prepared by the same general procedure by Fischer and Libowitzky,[10] although in this study an artificial coprohemin was used and an analogous coprobilirubin was isolated. The results, nevertheless, are just as significant so far as the underlying principle is concerned. This method of preparing bilirubin from hemoglobin in vitro has been confirmed by others.[11-13] The intermediary green compound differs from hemoglobin or hemin, as the case may be, through an opening of the protoporphyrin ring by an oxidative removal of the *a*-methene bridge. There is some reason to believe that the iron must be present for the porphyrin ring to open, at least under natural conditions. Thus, the administration of iron-free protoporphyrin to dogs with biliary renal fistulas was not followed by an increased excretion of bilirubin.[14]

It is probable that the so-called "pseudohemoglobin" of Barkan[15] is closely related to Lemberg's verdohemoglobin. (As prepared in vitro by the action of hydrogen peroxide, the compound obtained, according to Lemberg,[6] is actually a ferrous denatured globin-cyancholchemochromogen.) Barkan has shown that the iron of the pseudohemoglobin is "easily split off" by dilute hydrochloric acid, whereas that of ordinary hemoglobin is unaffected. The presence of a certain amount of easily-split-off iron in the hemoglobin of normally circulating erythrocytes indicates that at least a fraction of this hemoglobin is of the pseudohemoglobin or verdohemoglobin type.[9] Barkan[15, 16] has further shown that sterile incubation of erythrocytes for six hours is followed by an increase of iron and bilirubin in the supernatant plasma, as well as pseudohemoglobin in the red cells. All this indicates an intracorpuscular degradation of hemoglobin. The magnitude of the total wastage of hemoglobin is unknown. Recent studies by Watson and Paine[17] have shown that the erythrocytes sequestered in the splenic pulp and thrown into the splenic vein after adrenalin is administered, particularly in cases of hemolytic jaundice, are comparatively poor in hemoglobin. This reduction in hemoglobin concentration was unexplained except on the basis of intracorpuscular degradation during the period that the cells were in the spleen. The associated increased spheroidocytosis and increased fragility of the splenic-vein erythrocytes, noted after adrenalin, may be fundamentally correlated phenomena. Thus, Fahreus[18] has shown that stasis of red cells in the spleen, or in vitro, results in increased spheroidicity, which he believes to be due to formation of lysolecithin as a result of the change in the interface relation between the red cells and the plasma. It is quite possible that all these phenomena are correlated with a reduction of catalase in the erythrocytes, which Engel[12, 13] has shown to be of importance in retarding the conversion of hemoglobin to verdohemoglobin. Under ordinary circumstances, free hemoglobin in the plasma is quickly converted to bilirubin,[1] so that it is quite obvious that a relatively high degree of protection from oxidation obtains within the cells. Whether this is entirely due to catalase or not is unknown. This question is referred to below in the consideration of "pentdyopent."

In the conversion of hemoglobin to bilirubin, the fate of the globin is quite unknown. Duesberg[19, 20] suggested that the globin is never separated and that the plasma bilirubin is in fact a bilirubinglobin. This possibility will be returned to shortly. It may be noted first that Duesberg's belief was based on his own and Bingold's[21] observations that hematin, quite in contrast with hemoglobin, did not appear to be converted into bilirubin in vivo. A series of experiments* carried out in this laboratory have failed to confirm this belief and in fact have provided abundant evidence that the conversion of hematin to bilirubin is just as quantitative as that of hemoglobin.

Studies of the nature of the direct and indirect van den Bergh reactions have recently thrown much light on the general problem of bilirubin and the serum proteins. The earlier view that the indirect reacting bilirubin was attached to the serum globulin[22] has been shown to be incorrect.

*Watson, C. J., Pass, I. L., and Schwartz, S. Unpublished data.

The studies of Bennhold,[23] Pedersen and Waldenström[24] and Coolidge[25] clearly reveal that both direct and indirect types are bound to the serum albumin. Bennhold[23] points out that if it were not for the attachment of bilirubin to the serum albumin, jaundice would undoubtedly be more intense than it is, since the concentration of bilirubin is regularly higher in the serum than in the tissues. The indirect reacting bilirubin, according to Coolidge,[25] is chemically bound (with valence bonds), whereas the prompt, direct reacting type is simply absorbed loosely.

If Duesberg's[19, 20] hypothesis is correct, it follows that globin is closely akin to, if not identical with, serum albumin. Pedersen and Waldenström[24] emphasize the close physical similarity, the main difference being in the isoelectric point of hemoglobin (pH, 7.0) and serum albumin (pH, 4.8). The two proteins have identical molecular weights and they are not separable in the ultracentrifuge.[24] It is at least obvious, at this stage of our knowledge of the problem, that the globin could well remain attached throughout the transition of hemoglobin to bilirubin, and could then be incorporated in the serum albumin fraction, still attached to bilirubin. In fact, this appears to be much more reasonable than to assume that the bilirubin and globin are separated only to have the bilirubin reattach itself to another and very similar protein. Here again, it is evident that further investigation is needed.

From the foregoing statements, it should not be assumed that the different mechanism of the indirect, as compared with the prompt direct van den Bergh reaction, has been proved to depend on the type of attachment to serum albumin. It appears that this is important, but it remains to be shown that purely chemical differences in the bilirubin molecule are not also of significance. The question of furan-ring formation, as suggested by Fowweather,[26] was considered at some length in an earlier review.[1] The most recent studies of the Fischer[27] school indicate the presence of a furan ring in the molecule. Davis and Sheard[28] have shown that a distinct difference exists in the ultraviolet absorption spectrums of the prompt direct and the indirect reacting types of bilirubin. Previous studies had indicated another physicochemical difference between the two types — that is, a difference in filtrability through semipermeable membranes. Recent studies by Gregory and Andersch[29] failed to confirm this supposed difference. Snapper and Bendien[30] have shown, however, that in vitro studies of the ultrafiltrability of bilirubin are rendered difficult of interpretation because of a number of variable experimental factors. Heilbrun and Hubbard[31] have recently called attention again to the chloroform solubility of the indirect reacting bilirubin in the serum. They recommend simple repeated extractions of the serum with chloroform, which is separated by centrifuging and is then compared with a standard potassium bichromate solution. This appears to be a useful procedure, especially when a photoelectric colorimeter is not available for employment of the Malloy-Evelyn[32] method.

Another interesting theory regarding the basis of the different diazo reactions has been suggested by Colangiuli[33]: mesobilirubin is responsible for the delayed or indirect reaction, bilirubin only for the prompt direct. This assumption merits further study. Mesobilirubin is intermediary between bilirubin and mesobilirubinogen, the latter being one of the urobilinogens. Mesobilirubin is lighter in color than bilirubin (yellow rather than orange) but in other respects behaves like it, giving the Gmelin color play with yellow nitric acid, as well as the diazo reaction, but not Ehrlich's aldehyde test. The occurrence of mesobilirubin in nature has not yet been proved. It is readily prepared from bilirubin by suitable reduction, and it is entirely conceivable that bilirubin might be reduced in part either in the blood stream or in the organs with resultant mesobilirubin formation. It has been shown, for example, that crystalline stercobilin injected intravenously is often reduced in part to stercobilinogen.[1]

The determination of serum bilirubin has become much simpler and more accurate because of the adaptation of the van den Bergh method to the Evelyn photoelectric colorimeter. The procedure described by Malloy and Evelyn[32] permits ready distinction of the direct and indirect reacting serum bilirubin. Unfortunately, however, they recommend reading the total direct reacting bilirubin only after thirty minutes. This, of course, combines the prompt and delayed types, which have entirely different significance. In the laboratory of the University of Minnesota Hospital, it has been customary for some time to take readings at one minute and again at fifteen minutes, the first being the prompt reacting bilirubin and the second the delayed type. The total is then determined by the Malloy-Evelyn method, the difference between this and the amount observed at fifteen minutes constituting the indirect fraction. The original van den Bergh method and many subsequent modifications of it undoubtedly gave low values for total bilirubin because of some loss on the protein precipitated by the alcohol. Malloy and Evelyn[32] found that the indirect reaction was just as complete with 50 per cent or even lower concentrations of alcohol and that the serum pro-

teins were not precipitated at this concentration if the serum was first diluted. The sensitivity of the Evelyn colorimeter is quite sufficient to permit this dilution.

The bilirubin tolerance test of liver function, as originally described by von Bergmann and Eilbott, was the subject of a critical study by Strasser,[34] who concluded that the method affords a very delicate means of testing the bilirubin excretory function of the liver in nonjaundiced persons. More recently, Weech and his associates[35] have suggested a bilirubin clearance test applicable as well to jaundiced patients, the rate of removal being evaluated as a velocity constant of excretion, a measure that these investigators believe to be unaffected by elevation of the basal plasma bilirubin concentration.

Bilirubin Derivatives of Clinical Importance

Biliverdin

This substance, also known as dehydrobilirubin because it is formed by dehydrogenation of bilirubin, comprises the pigment fraction of verdohemoglobin, as noted above. There is no reason to believe, however, that biliverdin as such is contributed to the serum from destroyed hemoglobin. If this were true, accumulation of biliverdin — in other words "biliverdin icterus" — might be expected in cases of retention jaundice, especially those of hemolytic type. The occurrence of biliverdin in the serum has been studied recently in this laboratory.* With the Evelyn photoelectric colorimeter, the serum biliverdin is readily determined by virtue of its relatively strong absorption in the red portion of the spectrum. This study is not completed, but the results so far obtained indicate that biliverdin does not accumulate in pure retention jaundice. In regurgitation jaundice, whatever the cause, some biliverdin has been noted regularly. Except in jaundice due to cancer, however, the serum biliverdin has not exceeded 1.0 mg. per 100 cc., in accord with the observation that outspoken biliverdin jaundice is most frequently due to cancer of the biliary tract.[36] A few marked exceptions have been noted in which the jaundice was of parenchymal type (subacute or prolonged hepatitis or cirrhosis).

A "green reaction" with Ehrlich's test for urobilinogen in the urine is encountered in some patients with marked bilirubinuria. This has been shown to be due to biliverdin formed by the action of sufficiently high nitrite concentration in the urine, and stabilized at the biliverdin stage by the *p*-dimethylaminobenzaldehyde of the reagent.[37]

*Watson, C. J., and Larson, E. Unpublished data.

Urobilinogens and Related Compounds

Historical facts. In a consideration of recent advances in the study of this group, it is desirable for purposes of orientation to refer briefly to some of the earlier literature. The term "urobilin" was first employed by Jaffe[38] in 1866 to describe an orange pigment, which he observed both in bile and in certain urines. Jaffe noted that this substance exhibited intense green fluorescence with zinc chloride and ammonia, as well as a strong absorption band in the region of 500 millimicrons. In 1871, Vanlair and Masius[39] found an entirely similar pigment in the feces. With appropriate caution, since they were uncertain of its identity with Jaffe's urobilin, they designated the fecal pigment as stercobilin. The description of these substances was confirmed, in the main, by MacMunn[40] in 1878. It must be noted, however, that MacMunn described a distinct, although slight, difference in position of the absorption band of the urobilin from bile as compared with that from urine. In 1901, Ehrlich[41] described a color reaction in the urine with concentrated hydrochloric acid and *p*-dimethylaminobenzaldehyde. Although he was ignorant of the substance giving this reaction, he was aware that it was relatively common in cases of liver disease and in certain anemias. Neubauer[42] was the first to show that the reaction was due to the presence of a chromogen of Jaffe's urobilin. This chromogen was further studied by le Nobel,[43] who named it "urobilinogen." As early as 1887, Friedrich von Müller[44] had shown that the formation of urobilin and its occurrence in the urine were dependent on the presence of bile in the intestinal tract, thus demonstrating simultaneously the enterogenous origin of urobilin and its enterohepatic circulation. This work was confirmed many years later by the extensive studies of McMaster and Elman.[45] The first concrete knowledge of the chemistry of this group of substances was provided by Fischer[46] in 1911. He reduced bilirubin with amalgam, and then isolated the resulting chromogen in crystalline form.[46] This was first designated as "hemibilirubin," since the yield was less than 50 per cent and it was thought that but half the bilirubin molecule had been converted into the crystalline chromogen. In the same year, the substance was isolated from urines rich in urobilinogen (strong Ehrlich reaction).[47] By 1914, Fischer[48] had shown that mesobilirubin was intermediary and that there was no evidence that half the bilirubin molecule was converted to some other compound during the reduction. The chromogen was therefore renamed "mesobilirubinogen." On exposure to light and air, this substance develops urobilin characteristics, and the Ehrlich

reaction gradually disappears. The transition can be hastened by mild oxidizing agents, such as iodine. This was employed by Schlesinger,[49] together with alcoholic zinc acetate, in what has become the best known qualitative test for urobilin (not urobilinogen).

Recent fundamental advances. For a number of years after the studies of the Fischer school, the question whether mesobilirubinogen was the sole urobilinogen remained unanswered. Was the stercobilin of the feces identical with the urobilin derived in vitro from mesobilirubinogen? Prior to 1932, many attempts[46, 50–52] to answer this question by isolating either chromogen or pigment from the feces had failed. In 1932, crystalline stercobilin was isolated from human feces.[53, 54] The same substance was obtained from pathologic human urines in 1933.[55, 56] A method was then devised for preparing crystalline urobilin from mesobilirubinogen, and it was shown that this urobilin differed in a number of respects from stercobilin.[57, 58] This was confirmed by Fischer and his co-workers.[59–61] Siedel and Meier[61] synthesized this compound and designated it as urobilin IX *a*, indicating simply that the configuration corresponds with that of the protoporphyrin in the hemoglobin molecule — that is, Type IX of the fifteen possible isomers — and also that the porphyrin ring has opened at the *a*-methene bridge. Fischer and his associates[59] found that although urobilin IX *a* is optically inactive, stercobilin is strongly levorotatory. This will be referred to again in the following. They also noted that the melting points of the free substances were distinctive when recrystallized from acetone.[60, 61] Shortly after the isolation of stercobilin, attempts were made to reduce it and isolate the chromogen to determine whether or not this was mesobilirubinogen. The reduction is easily carried out, but the resulting chromogen (stercobilinogen) has not yet been crystallized, although mesobilirubinogen crystallizes readily in much smaller amounts under the same conditions. Recent studies have to some extent clarified the nature of stercobilin and its relation to mesobilirubinogen.

The possibility that there were two bilirubins in the bile — one on reduction in the intestinal tract yielding mesobilirubinogen and the other stercobilinogen — was first considered. It was quite conceivable, for example, that some of the porphyrin of the hemoglobin molecule might open its ring, not at the *a*-methene bridge, but at one of the other three. This, of course, would result in the formation of a different bilirubin, or a bilirubinoid substance. With this in mind, a number of human bile samples were reduced directly with amalgam, until the Ehrlich reaction had become strong.[62] The resulting urobilinogen, after partial purification, was converted to urobilin, which was then isolated. In every sample, this was urobilin IX *a* on the basis of optical activity, absorption spectrum and melting points. Stercobilin was never formed in this way, although it was occasionally native* in the bile, in which event it was first removed. This study revealed conclusively that the formation of stercobilin is dependent on a more complex sequence of events than the simple reduction of the bilirubin present in the bile. Lemberg[63] recently recommended that the term "stercobilin" be abandoned, but in view of these findings and other observations, which will be mentioned, it appears that the term is more descriptive than any other, since it is now certain that stercobilin formation is dependent on a peculiar chemical activity in the intestinal tract, probably related intimately to bacterial metabolism in the colon.

Experiments were next carried out with the specific purpose of determining whether stercobilin is derived from mesobilirubinogen. The possibility existed, of course, that it might be an offshoot from any of the intermediary reduction compounds in the pathway from bilirubin to mesobilirubinogen, such as mesobilirubin and dihydromesobilirubin. It was found that if crystalline mesobilirubinogen was given by mouth this was followed by an increased output of stercobilin in the feces.[64] Incubation of mesobilirubinogen with normal feces resulted in a clear-cut increase of stercobilin.[64] This was not true, however, of strictly acholic feces, in which mesobilirubinogen was unchanged. The addition of bile to such samples caused stercobilin formation in some, whereas a type of urobilin that is regularly observed in infected bile samples appeared in others. This new or third urobilin, as described by Schwartz and Watson,[65] is of particular interest in that it is dextrorotatory in the same magnitude that stercobilin is levorotatory. The melting point is the same as that of stercobilin, but the absorption is identical with that of urobilin IX *a*. The first two characteristics suggest that *d*-urobilin, as this substance has been designated tentatively, may be a stereoisomer of stercobilin. Further studies of this question are planned. Dextro-urobilin is further characterized by a color play with dioxan hydrochloric acid.[65] A slight difference in the absorption maximums of urobilin IX *a* and stercobilin was first reported by Lemberg,[66] although the former had not at that time been crystallized or identified as

*The presence of stercobilin in the bile may be ascribed without doubt to the enterohepatic circulation. Crystalline stercobilin, when injected intravenously, reappears in the bile unchanged.

a distinct entity. Dextro-urobilin may usually be obtained from infected human fistula bile, although the yields are small owing to the difficulty of purification. Schwartz, Sborov and Watson[67] have further noted that mesobilirubinogen added to such infected samples is at least partially converted to *d*-urobilin. The significance of this change, as compared with that to stercobilin in normal feces, is as yet unknown. The above-mentioned experiments with acholic feces, bile and mesobilirubinogen, in which on one occasion stercobilin and on another *d*-urobilin were formed, indicate that some unknown factor influences the direction of the conversion. It is also unknown whether there is a separate chromogen for *d*-urobilin as there certainly is for stercobilin, or whether mesobilirubinogen, under proper conditions, is converted directly to the dextrorotatory compound. As yet, no clinical importance has attached to this substance. It is quite conceivable that it will be found in the urine in biliary-tract infections, since it is well known that urobilin may be formed in the infected biliary tract.[45] It would also be of interest to investigate the bile obtained by duodenal drainage, with respect to the presence of *d*-urobilin. There can now be little doubt that the slight spectroscopic difference recorded for bile urobilin by MacMunn,[40] in 1878, was due to the presence of *d*-urobilin.

As a result of these studies, it is evident that urobilinogen as tested for by the Ehrlich reaction is not a single substance. Actually, there are at least two urobilinogens,—mesobilirubinogen and stercobilinogen,—and it remains to be determined whether *d*-urobilin has a separate chromogen or is derived from mesobilirubinogen directly. The relation of these various derivatives obtained by reduction of bilirubin is shown in Figure 1.

Thus, the quantitative determination of urobilinogen in the urine and feces includes any or all of the chromogens present in the sample. It has been shown repeatedly that both stercobilinogen and mesobilirubinogen occur in the feces and urine.[1] There is no evidence at present that either of these substances has a different clinical significance from the other. Lemberg[63] suggested such a possibility on the basis of a differential test for stercobilin and urobilin IX *a* that was applicable to urine extracts. He found that heating with iron chloride and hydrochloric acid produced a violet reaction with urobilin IX *a*, whereas stercobilin was unchanged. When this method was applied to urine samples from various diseases, stercobilin was regularly found, and urobilin IX *a* but rarely. Lemberg therefore suggested that the latter might be pathologic. On the other hand, mesobilirubinogen (the precursor of urobilin IX *a*) has been isolated from the urine in considerable amount in widely divergent conditions, such as hepatic cirrhosis and other diseases,[46, 48] including hemolytic jaundice.[68] In hemolytic jaundice, stercobilin was obtained from the mother liquor in an amount of the same magnitude as that of the mesobilirubinogen. In

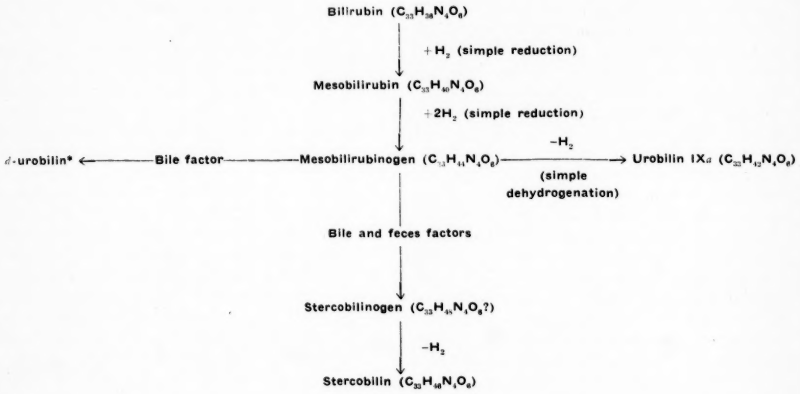

FIGURE 1.

*As yet there have been no elementary analyses of this substance, so that the empirical formula is unknown.

a consideration of Lemberg's suggestion, the question must be taken into account of the relative amount of stercobilinogen formed in the colon as compared with that of unchanged mesobilirubinogen. Exact evidence on this point is lacking. Nevertheless, it will be seen in Figure 1 that when bile is entering the intestinal tract, the factors are present for the conversion of mesobilirubinogen to stercobilinogen. Quite in accord with this belief, it has been noted during numerous isolations of crystalline stercobilin from various samples of feces, that this substance appears to predominate in amount over the remaining fraction of the urobilin that is not crystallized (some of which is probably urobilin IX *a*). It is true that a comparison of amount is rendered somewhat difficult because mesobilirubinogen and urobilin IX *a* are not so stable as stercobilinogen or stercobilin and that they are converted in part to mesobiliviolin during the isolation procedure.[56, 70] In spite of this, it is believed that the majority of the bilirubin of the bile is eventually excreted in the feces as stercobilinogen and that any marked preponderance of mesobilirubinogen in a given urine sample indicates either an unusually rapid absorption of mesobilinogen from the right colon or an altered chemical activity in the colon—for example, diminution of the factors necessary for the conversion of mesobilirubinogen to stercobilinogen.

(To be concluded)

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NEW HAMPSHIRE MEDICAL SOCIETY

PROCEEDINGS OF THE ONE HUNDRED AND FIFTY-FIRST ANNUAL MEETING

May 12 and 13, 1942

Tuesday, May 12

THE scientific session of the New Hampshire Medical Society was opened at the Hotel Carpenter, Manchester, at eleven-thirty o'clock in the morning, with President Charles H. Dolloff presiding.

Dr. Deering G. Smith made the following remarks on the Procurement and Assignment Service.

I want to thank the members of the Society, especially the members of the county and state Medical Preparedness and Procurement and Assignment Committee, for the excellent work they have been doing. I expect that this co-operation and work will continue.

The Procurement and Assignment Service has been established to decide which doctors will go into the Army or Navy and which doctors will be considered essential for the care of the civilian population. In other words, at the same time the Army needs are taken care of, sufficient doctors must be kept at home to take care of civilian needs.

It has been stated that the Selective Service System is the dogcatcher, and the Procurement and Assignment Service is the Society for the Prevention of Cruelty to Animals.

In this state, the organization is headed by myself, as chairman; serving on the committee are Drs. Jones, of Manchester, and Bowler, of Hanover. In each county, there is a similar committee to advise the state committee.

Medical-enrollment forms have been sent to each doctor, and should be filled out and returned to the proper authority at once. If any of the doctors have not received these blanks, I shall be glad to furnish one.

Most of the doctors in the state have been classified as 3*A* under the Selective Service System. General Bowen, head of the Selective Service System in New Hampshire, has received instructions, which he is sending to the local boards today or tomorrow, notifying them to reclassify the doctors who are now in 3*A*. In the reclassification, the amount these doctors will receive as commissioned officers in the Army and Navy will be considered in figuring out whether or not they have dependents in sufficient number so that they will have to stay at home, rather than go into the Army.

The belief is that practically every doctor will be reclassified from 3*A* to 1*A*. He will be given a reasonable time to apply for a commission in the Army if he is not considered essential, and will then, if he does not apply for a commission, be inducted as a private.

The need for medical officers is 15,000 more in 1942, and 5000 before July 1. That breaks down to 50 the number of medical officers needed in New Hampshire before July 1. To assist in the securing of this quota, the National Recruiting Board has established a medical recruiting board in New Hampshire. That is headed by Captain Weimars, of the Medical Corps. It gives me pleasure to introduce Captain Weimars.

Captain Weimars spoke as follows:

The Medical Officers' Recruiting Board will probably be located at 77 Main Street, Nashua. The purpose of this organization, as Dr. Smith has pointed out, is to get medical officers for the Army. He has told you how many are needed — about 5000 from the United States to be obtained before July 1. And, of course, an applicant must be physically fit before he can be given a commission.

We have the authority to cut the red tape, or at least to make it as light as possible, to get medical officers into the Army. We have the authority to act for the Surgeon General. We have the authority to disqualify or qualify physically any applicant for medical duty.

If you have been reading the *Journal of the American Medical Association*, you will have noted that the physical qualifications for medical officers have been considerably reduced. We have now what is termed a limited service. The limited service covers everything but field duty. Probably, such men will not see foreign duty. Thus, we can waive certain physical defects, such as missing teeth, an insufficient number of teeth, carious teeth and other minor defects.

An applicant must be a licensed practitioner of the State, must have had one year's internship, must be a citizen of the United States, and must be between the ages of twenty-six and forty-five. We can offer commissions to any person under thirty-seven to rank as a first lieutenant. We are authorized to recruit and commission applicants as first lieutenants, unless they have special qualifications, are members of a specialty Board or fellows of the American College of Surgeons or the American College of Physicians.

However, we have the authority, if applicants over thirty-seven and under forty-five meet certain qualifications, to commission them as captains.

A first lieutenant, unmarried and without dependents, gets approximately $2700 a year. A first lieutenant, married, or having dependents, gets approximately $3100 a year. A captain, unmarried, gets $3300 a year, and a captain, married, gets $3800 a year.

We have on our medical recruiting board a member from the Office of the Adjutant General, who has the authority to swear in an applicant and give him his commission, serial number and rank and have him ready for active duty within a very short time. Accordingly, I present to you Lieutenant Veagle.

Lieutenant Veagle spoke as follows:

The Army needs medical officers. That is naturally true because a good many of your fellow citizens have been taken into the armed forces; consequently, some of you will have to go along with them to help protect them and keep them in good health and bring them back again. This program is being speeded up. Heretofore, to obtain a commission, it took weeks and months and a good deal of red tape; the necessary papers were incomplete, or lost here and there in Washington. When we once start to operate, a physician probably will be able to obtain a commission within a week at the most. Of course, you will be allowed fourteen days after you receive your commission, or are sworn in, and under certain conditions you will be allowed a greater length of time. If you wish to waive the fourteen days and immediately go on duty, undoubtedly we can find an assignment for you within a few hours.

Dr. Metcalf read the following letter from the Bureau of Narcotics at Boston:

I deem it necessary to bring to the attention of the officers of the New Hampshire Medical Society information that should be disseminated for the protection of its members.

About this time of year, numerous carnivals, circus companies and outdoor shows start their season and visit various cities and towns throughout the State. This bureau has learned from experience that some of the personnel of these groups and their followers are frequently drug addicts. To obtain narcotics for the satisfaction of their addiction, these persons approach physicians in the towns where they are appearing, usually with a convincing story of an ailment that requires morphine for relief; or even a story that they are on the way to a sanatorium for treatment and need a certain amount to tide them over. Physicians cannot be too careful about being prevailed on by these people, who have no medical need for narcotics.

I strongly urge the members of your society to be on their guard against the danger of being imposed on by such addicts. Unfortunately, the New Hampshire state law is inadequate in its provisions for prosecution of addicts who obtain narcotics, for the satisfaction of their addiction, under pretense; and the federal law has no punishment for the addict. However, under the federal law, any physician who prescribes or dispenses drugs indiscriminately, other than for an established medical need, is subject to prosecution for violation of the Harrison Narcotic Act, as amended. Every physician is required to determine, from a personal examination of a patient, that patient's medical need for narcotic drugs before prescribing or dispensing same. A physician should never accept the word of a patient that he has a certain physical ailment and requires narcotics for relief, or that a physician in another city has been giving him prescriptions for a certain amount. You are cautioned to exercise extreme care to prescribe and dispense narcotics in the course of your professional practice only, and to patients under your care whose medical need has been definitely established by yourself.

In these days when careful conservation of the supply of narcotic drugs on hand is necessary because of world conditions, the Bureau of Narcotics is confident that the medical profession will co-operate wholeheartedly to prevent any diversion of these drugs to illegitimate channels.

WM. E. CLARK, *District Supervisor*

The morning session was concluded with an address by Dr. Paul D. White, of Boston, who opened the symposium on heart disease with a paper entitled "Important Clues in Cardiovascular Diagnosis."

The afternoon session convened at two o'clock, with President Dolloff presiding.

Dr. Ashton Graybiel, of Boston, continued the symposium on heart disease with a paper, entitled "Electrocardiography." Dr. Richard Schatzki, of Boston, concluded the symposium with an address, "Cardiovascular X-ray."

Dr. Beatrice M. Kesten, of New York City, then presented a paper, entitled "Dermatology." The afternoon session was concluded with a talking, motion-picture film in color, entitled "Sex Hormones: Physiology, diagnosis and therapy," which was presented by Parke, Davis and Company.

WEDNESDAY, MAY 13

The morning session convened at eleven-fifteen o'clock, with President Dolloff presiding.

The following remarks were made by the visiting delegates from other New England state medical societies:

DR. CARL E. RICHARDS, Alfred, Maine: It gives me great pleasure to be able to attend this annual meeting of the New Hampshire Medical Society, and to be able to extend the greetings of the Maine Medical Association to you. I have enjoyed the meeting very much up to this point, and I think the papers and discussion groups have been above par.

Being a general practitioner, I was particularly impressed with the papers presented yesterday in the symposium on heart disease and the paper on dermatology by Dr. Kesten.

I am looking forward to hearing the papers by Drs. Irons, Adair and Barker, and also to the banquet tonight, which I believe is a standing event.

The annual meeting of the Maine Medical Association will be held at the Poland Spring House, Poland Spring, Maine, on June 21, 22 and 23, and I have been asked by the president of the association to extend an invitation to the members of the New Hampshire Medical Society. The way things are with gasoline and tires and the like, we believe that this may be one of our last large meetings for the duration, and an outstanding program has therefore been arranged, which will be brought to a conclusion by the appearance of Dr. Morris Fishbein at the annual banquet on Tuesday evening, June 23.

DR. BERTRAND F. ANDREWS, Middlebury, Vermont: I was able to attend your meeting only today, so that I have not been here throughout the whole meeting. However, I am very glad to be able to be here with you and to bring you the greetings of the Vermont State Medical Society.

DR. C. BERTRAM GAY, Fitchburg, Massachusetts: I am very glad to be with you, and to bring the greetings of the Massachusetts Medical Society, as well as greetings from our president, Dr. Ober.

I came in a little while ago, and I see that you have a very interesting gathering here. Earlier this morning, I attended the Surgical Section, and heard a good discussion of gall-bladder disease.

We invite you to be with us at our annual meeting in Boston at the Hotel Statler in two weeks.

DR. JOHN J. CURLEY, Leominster, Massachusetts: I think that Dr. Gay has expressed the thoughts of the Massachusetts Medical Society very well.

DR. PAUL R. FELT, Middletown, Connecticut: By way of a long-continued and friendly tradition, I extend to you the greetings of the Connecticut State Medical Society. I can even say that after an abominable bus ride from Middletown to Manchester! We cordially invite all of you to the annual meeting of the Connecticut State Medical Society in Middletown the first week in June — I am not exactly sure of the dates at the moment. Our meetings will be held on the campus of Wesleyan University, and we are planning a very enjoyable time and shall be glad to see you there.

Dr. Ernest E. Irons, secretary of the Board of Trustees of the American Medical Association, presented the first paper of the morning, entitled "Aspiration Pneumonia." This concluded the morning session.

The afternoon session convened at two o'clock, with President Dolloff presiding.

Dr. Zatae L. Straw, of Manchester, was awarded a fifty-year-membership gold medal, and spoke as follows:

I thank you very sincerely for this signal honor, which you have conferred on me today in giving me this medal as a token of fifty years of busy, interesting and, I hope, worthy service in the practice of medicine in New Hampshire.

Of course, my being here is due to two factors only. One is the magnificent health I inherited from a long line of stalwart ancestors; the other is a determined and, I might say, congenital vocation to study medicine, because in my immediate family there were for years eight practicing doctors, although at present there are only five in three different states — New Jersey, Pennsylvania and New Hampshire.

I know the secret of popular public speaking. This is the recipe, which I intend to follow: be brief; be sincere; be seated. I shall not detain you for more than a moment, nor am I going to subject you to any résumé of the wonderful progress in the medical field during these fifty years. But I must refer, briefly, to the fact that, when I began to practice, we did not have sulfanilamide or the wonderful group of derivatives in that family. We did not know anything about insulin, through which years of comfort and active life are added to an otherwise hopeless outlook, or plastic and orthopedic surgery and the wonder-working feats that are performed today, through which mangled and hopeless wrecks of humanity are transformed into active citizens. We had none of the modern anesthetists and the agents that they work with. We had practically no antisepsis or asepsis to speak of, as compared with what we have today. We knew nothing about blood transfusions or serum therapy. We did not know much of anything, some of you younger chaps will think, but we got along, and sometimes our patients got well. We are grateful. Once in a while they paid us, and that was a help, too, even then. If they did, it was all right, because that old doctor was not only the physician but also the legal adviser and minister and friend and moneylender and a good neighbor in every sense of the word.

Nowadays, we have no time for that. But that is the way it used to be.

We hope for much better things. However, in the next fifty years, I cannot possibly foresee such great progress in this or any other field as we have experienced in the last fifty years.

It has been a great treat, a great boon, to have lived in these fifty years, and I doubt if the next hundred years will hold as much interest and progress in any line.

Fifty years ago, the woman doctor was an unknown quantity, more or less, a rare species. They hardly knew how to take us. But, I joined the medical society, and since that time I have been accepted and treated just like one of you, and that is precisely as I wanted to have it. I have had a square deal in every way, and I want to express my appreciation and gratitude to the boys in Manchester, to my associates in the medical profession in Manchester, for their unvarying courtesy and friendly, professional consideration during all these years.

Another fifty-year-membership gold medal was awarded to Dr. Edwin P. Hodgdon, of Lakeport, who spoke as follows:

I thank the members of the New Hampshire Medical Society for this wonderful emblem. I never really expected to receive one, because twenty-five years ago, I was refused life insurance by a well-known company because of my family history and also because of a large abdominal girth. However, I served notice on the Chief Medical Examiner that I would be present at his funeral!

There is an old saying that the first fifty years are the hardest, but I want to rebut that statement: I can truthfully say that the first fifty years have been grateful ones to me. In the "Gay Nineties" I think I enjoyed myself as much or more than I have in the following years. We did not have tire or gasoline troubles. The only thing we had to find out was the price of plain oats.

Dr. H. O. Smith then made the following remarks:

In September, 1859, a baby boy appeared in the town of Milford, and his parents burdened him with the long name of Herbert Stillman Hutchinson. The boy waxed in stature and in wisdom. In due time, he entered Dartmouth. He graduated from Dartmouth in the year 1875, sixty-five long years ago. Today, he is the oldest in the list of living Dartmouth graduates.

After graduation, he began the study of medicine. He went to Bellevue Hospital Medical College, which did itself honor by giving him the degree of M.D. in 1880.

After his graduation, he returned to his home town of Milford, and he has been practicing medicine there ever

since. He is known and loved by every man, woman and child in that town.

Ten years ago, we gave Dr. Hutchinson a gold medal representing his fifty years of membership in the Society. Today, he has attained the rare distinction of having been a member for sixty years.

Mr. President and members of the Society, I deem it a great honor and a great privilege to present to you Dr. Herbert Stillman Hutchinson, our sixty-year member.

Dr. Hutchinson spoke as follows:

It was a considerable surprise to me when I was asked to be here today. I had supposed that my presence was no longer needed.

The two members of the Society who have preceded me and I belong to what is known as the horse-and-buggy days, and that means more to us, perhaps, than to those who have gone into the service more recently: bad roads, and more nightwork than is now indulged in. It meant muddy roads and ruts, spring and fall, and deep snows and unbroken roads in the wintertime. It meant everything that was exhausting to a person, if he so willed to take up the duties that he was called on to perform.

Since then, many things have happened. Many have passed on. The country doctor no longer has such a hard time in getting from one place to another. The automobile and better roads have revolutionized in a great measure the hardships that we formerly experienced.

After four years in the practice of medicine in Francestown, New Hampshire, I moved to Milford, for several reasons. One was that the work was so hard that I could not stand it; I was quite rugged, too. The other was that my father, the owner of a great farm on which I was born and brought up, was very ill, and I thought that it was my duty to be where I could help him. I am getting along in years and most of my friends and associates and co-workers in medicine and surgery have passed on.

An eminent lawyer in a neighboring town said to me one day: "Doctor, ever since I was fifty years old, I feel that I have been occupying most of my time burying my friends." And that really occurs to me very often. Although the life span has increased and people live to be older, there are just as many dying as ever did, but this comes in the old-age variety of disease, which makes it a rather hopeless task to attempt to effect a lasting cure.

Medicine is hard work. We meet much prejudice, and we meet people with set ways, who are unwilling, sometimes, to abandon their notions of remedies and to follow our desired prescriptions. But we keep busy, and that is the answer to it all.

Being somewhat aged myself, I am now pretty well able to experience some of the symptoms that our patients display, and to know whether or not they are telling the truth. It is a human frailty to exaggerate sometimes and to misrepresent intentionally or otherwise, the symptoms, as well as to minimize them, and to refuse to follow set directions, and that constitutes one of the hardest obstacles in the practice of medicine. If a person will not cooperate it makes an incomplete piece of work, and our work is hard enough as it is, without these obstacles.

An ex-president of the Society, not many months ago, made this remark to me: "Doctor, do you realize the difference in the preparation of the graduate from the medical school of today and what it was when you were graduated?" I said to him: "My dear man, I have recognized that every moment of my life." We did not have the facilities presented to us to round out our courses and make things easy that are presented today. And, today, not only are they presented free but they are required. That is true in obstetrics, and it is true in surgical technic and various other things, and it is a great advantage.

And my ex-president friend continued: "When the doctors nowadays start out, they are several laps ahead of you fellows when you started. Do you realize that?" And I said: "I certainly do." It keeps me busy reading and studying and trying to keep abreast of the times. But it is work, and it is work that I love and that I shall follow so long as I have the strength to do so. There is no better remedy for the blues or for discouragement than hard work. Since this is my chosen occupation in life, it will follow with me to the end.

Major Weir, in the absence of General Bowen, spoke as follows:

General Bowen called me up a short time ago, announcing that he could not come here, but he did want me to express to the members of the Society his sincere thanks for the co-operation shown to him during the past year. He said that without that co-operation the Selective Service System could not have functioned at all smoothly, and from my own point of view, I want to thank all the physicians of the Society who have been in selective-service work for their co-operation with me.

I think we can say that the record of the Society is unmatched, in that I do not believe there has been one resignation from the system, except for illness, or moving out of the State.

Secretary Metcalf then read the following resolution concerning the resignation of Dr. Henry O. Smith as a trustee.

> WHEREAS, the House of Delegates learns with regret of Dr. Henry O. Smith's desire to retire from the Board of Trustees,
>
> THEREFORE, BE IT RESOLVED, That the House express to Dr. Smith its deep appreciation of his outstanding service to the New Hampshire Medical Society in the promotion of its best interest through his wise counsel and active participation in all its affairs during the many years of his membership.

Dr. Metcalf continued as follows:

The House of Delegates directed the Committee on Child Health to co-operate with the appropriate state and federal agencies to immunize, against diphtheria and smallpox, all children over nine months of age.

It awarded $50 to the Cancer Committee to carry on its work for the coming year.

It decided to be a cosponsor in the New England Health Institute, which meets in New Hampshire in 1943. Details of this co-operation were left to the Committee on Public Health.

It accepted a bill concerning medical-service corporations drawn up by Mr. Sulloway, and directed me to submit it to the Committee on Public Relations, who will submit it to the Legislature next year for their consideration.

The House of Delegates decided to co-operate with the National Physicians' Committee, and it requests the members of each county society to contribute $1.00 apiece. The Committee on Public Relations is to co-operate with the

National Physicians' Committee, the county secretary to turn the money over to the Treasurer, to be forwarded. That is for the expenses of the National Physicians' Committee.

It directed the Committee on Public Health to cooperate with the State Board of Health, to determine the minimum requirements for first-aid rooms in industry.

It directed me to write to our senators and representatives about a bill in Washington that would permit osteopaths to serve in the United States Army hospitals.

It directed me to notify Congress that they were opposed to a change in Section I of the Revenue Laws, which would permit the taxation of charitable and educational institutions.

It decided to cover the cost of the Procurement and Assignment Service from the general funds of the Society, so that we may run that service and not have it run by a federal agency.

It decided that residence in New Hampshire is not necessary for membership in the Society. Two or three men who formerly lived here and who now live outside the State wish to continue as members.

The House of Delegates decided to come to Manchester again in 1943. The meeting will presumably be held on May 11 and 12. You will be glad to know that I have already started to work on the 1943 program.

Your new vice-president is Dr. James W. Jameson, of Concord. Your new president is Dr. Timothy F. Rock, of Nashua. Dr. Sanders, of Claremont, was elected vice-president last year, and would naturally have moved up to the presidency. He said that he was unable to carry on the work of the presidency, so that Dr. Rock was elected in his place.

Dr. H. O. Smith then presented the report of the Trustees.

FINANCIAL REPORT OF THE TRUSTEES OF THE NEW HAMPSHIRE MEDICAL SOCIETY FOR THE YEAR ENDING JANUARY 1, 1942

Receipts

Interests on various deposits, other than Benevolence Fund	$317.12

Expenditures

Expenses of meeting (Sesquicentennial) 1941	1,000.00
Pray and Burnham prizes	100.00
Total expenditures	1,100.00
Decrease in funds, other than Benevolence Fund	782.78

GENERAL FUND

Deposits: New Hampshire Savings Bank	$4,859.13
Portsmouth Trust and Guarantee Company	2,005.46
Nashua Trust Company	205.18
United States Defense Bonds, Series "G"	3,000.00
Total funds available on January 1, 1942	$10,069.77

BARTLETT FUND

Deposit: Portsmouth Savings Bank ($352.11 of this is a permanent fund, the income to be "expended only for the benefit of medical science, as may be directed by vote of this society")	$2,718.19
United States Defense Bonds, Series "G"	2,000.00
Total funds	$4,718.19

PRAY FUND

Deposit: Strafford Savings Bank ($1000 of this is a permanent fund, the income to be expended only for prize essays)	$217.41
United States Defense Bonds, Series "G"	1,000.00
Total	$1,217.41

BURNHAM FUND

Deposit: New Hampshire Savings Bank ($1140 of this is a permanent fund, the income to be expended only for prize essays)	$1,237.32
United States Defense Bonds, Series "G"	1,000.00
Total	$2,237.32

BENEVOLENCE FUND

Deposit: New Hampshire Savings Bank ($394.50 of this is accrued income available for the purposes of the fund)	$1,385.69
United States Defense Bonds, Series "G"	3,000.00
Total	$4,385.69

A prize of $100.00 was awarded for a paper entitled "Dermatotherapy as Applied to Diseases of the Eyelids" by "Eidal," pseudonym for Dr. Arthur Linksz, and also a prize of $50.00 for a paper entitled "Prostatic Hypertrophy" by "John H. Doe," pseudonym for Dr. Rolf Lium.

The accounts of the Treasurer have been examined and found correctly cast and properly vouched.

HENRY O. SMITH
SAMUEL T. LADD
FREDERIC P. LORD, *Secretary*

The scientific session was concluded by an address, entitled "Recent Treatment of Gonorrheal Infections in the Female," which was presented by Dr. Fred L. Adair, of Chicago.

The banquet was held at the Hotel Carpenter, with Dr. Ralph W. Tuttle as toastmaster. President-Elect Timothy F. Rock, of Nashua, and Governor Robert O. Blood spoke very briefly. Dr. Ernest E. Lyons then presented an address, entitled "The Last Illness of Sir Joshua Reynolds," and showed many lantern slides of paintings. This was followed by a talk by President Dolloff.

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

CASE 28441

PRESENTATION OF CASE

First admission. A fifty-two-year-old truck driver came to the hospital because of swelling of the abdomen.

Five years prior to admission the patient noticed that his abdomen was becoming swollen. Three years later he noticed a feeling of abdominal fullness that developed approximately twenty minutes after eating. At times, especially when constipated, this was accompanied by pain on both sides of the abdomen at about the level of the umbilicus. These symptoms were relieved by soda. At the same time he noticed the appearance of a yellowish discoloration of the skin, unaccompanied by nausea, vomiting, pain or clay-colored stools. He also developed exertional dyspnea and ankle edema. One year before entry, urinary urgency, frequency, hesitancy and dribbling and nocturia developed. These symptoms became progressively worse to the day of admission. There was no loss in weight or any blood in his urine or stools. For many years he had occasionally drunk 3 or 4 ounces of whisky each day, but usually it was less; however, his diet was said to have been quite adequate.

His wife and two children were living and well. His wife had had no miscarriages.

The patient was born in Italy and came to America at the age of three. His vision had recently become poor, but there was no double vision, blurring or spots before the eyes. At the age of eighteen or nineteen, he had had gonorrhea without known sequelae. He denied a penile sore.

Physical examination revealed a heavy-set man who appeared slightly dyspneic. The skin and scleras were obviously yellow. The abdomen was "massive," tense and bulging. There was a marked wasting of the soft parts of the face, arms and chest. No odor of acetamide was detected on the breath. There was a marked degree of clubbing of the fingers and toes. The chest was barrel shaped. Examination of the lungs was negative except for bilateral posterior basal rales. The heart was not enlarged. A loud systolic murmur was audible over the entire precordium but best heard in the fourth interspace to the left of the sternum. A fluid wave was easily elicited, and there was shifting dullness. Rectal examination revealed internal and external hemorrhoids. The prostate was nodular and not tender. There was edema of both lower extremities.

The blood pressure was 144 systolic, 60 diastolic. The temperature was 98° F., the pulse 81, and the respirations 20.

Examination of the blood revealed a hematocrit of 17.0 per cent, a red-cell count of 2,250,000, with a hemoglobin of 22 per cent, and a white-cell count of 2300 with 71 per cent polymorphonuclears. In the blood smear there was extreme variation in the size and shape of the red cells. Many macrocytes were present, but the average size of the red cells was below normal; there was no polychromatophilia; the granulocytes appeared old. The urine had a specific gravity of 1.015, and the sediment contained 2 white cells, a rare red cell and occasional epithelial cells and coarse granular casts per high-power field. The blood Hinton and Wassermann tests were positive. The van den Bergh test was first reported normal; when repeated four days later it was said to have been slightly above normal with a biphasic reaction. The blood protein was 5.4 gm. per 100 cc. The prothrombin time was 22 seconds (normal, 20 seconds).

Roentgenograms of the chest showed slight generalized cardiac enlargement. The aorta was not remarkable, and the lung fields were clear. An abdominal paracentesis yielded 5000 cc. of clear fluid with a specific gravity of 1.012 and a cell count of 900. The fluid did not clot on standing. The spinal fluid was essentially normal; the total protein content was 30 mg. per 100 cc., the colloidal gold curve 0012211000, and the Wassermann test negative.

After paracentesis an irregular mass was felt below the costal margin, which was believed to be the liver. On the seventh day after admission, under local anesthesia, a peritoneoscopy was performed; 6500 cc. of straw-colored fluid was aspirated. The liver could not be seen anywhere because thick masses of omentum were adherent to it over both lobes. On the left the omentum was also adherent to the anterior abdominal wall and was very vascular suggesting a spontaneous omentopexy. From the general point of attachment of the omentum from inspection and from percussion after removal of the fluid, it did not appear that the liver was enlarged. The lower abdomen and pelvis appeared normal. The patient received supportive and antisyphilitic therapy, including potassium iodide and bismuth subsalicylate, and was discharged moderately improved on the twelfth day.

Second admission (seven months later). Following discharge, the patient did poorly. Ascites reaccumulated rapidly, and during the month prior to admission required a tap every four to six days, when 4000 to 5000 cc. of fluid was recovered. Intermittent jaundice had been observed in the Out Patient Department. The potassium iodide and bismuth subsalicylate had been continued. The patient's appetite had been fair, although he severely limited his fluid intake. On the day of admission he developed a dull aching pain beneath the right costal margin.

Physical examination revealed a thin, emaciated dyspneic man who was obviously acutely and chronically ill. The skin was dry, yellow and sallow. There were multiple port-wine telangiectases over the hands and forearms. Expansion of the chest was limited. There were diminished to absent breath sounds over this area with decreased tactile and vocal fremitus. There was flatness to the level of the fifth interspace posteriorly and laterally. Multiple coarse rhonchi were audible in both chests. The heart was not enlarged. There was a great deal of abdominal fluid. After withdrawal of 6000 cc. of fluid the liver edge was felt four fingerbreadths below the costal margin; the edge was rough and not tender. The abdominal-wall veins were prominent. A mass could be felt in the left upper quadrant. An umbilical venous hum was audible. There was a moderate pitting edema of the legs and abdominal wall.

The blood pressure was 120 systolic, 60 diastolic. The temperature was 98°F., the pulse 82, and the respirations 20.

Examination of the blood revealed a red-cell count of 1,920,000 with a hemoglobin of 3.2 gm. and a white-cell count of 10,600 with 78 per cent polymorphonuclears. The smear resembled that of the first admission. The urine and stool examinations were negative. The blood protein was 5.6 gm. per 100 cc. An x-ray film of the chest revealed extensive increased density involving the lower two thirds of the left lung field, more marked laterally. The heart was about normal in position. Abdominal paracentesis was repeated several times, but the fluid reaccumulated rapidly. A thoracentesis yielded 1100 cc. of dark-amber, slightly cloudy fluid. This did not represent all the chest fluid, since dullness was still present to the fifth interspace.

Terminally, the patient was very uncomfortable because of dyspnea. Hoarseness developed, and the patient talked with a whisper. Examination of the larynx revealed a moderate edema of the arytenoid cartilages, which somewhat limited the excursion of the true cords; the airway was perfectly adequate. The patient became drowsy and stuporous, and died on the sixteenth day after admission.

Differential Diagnosis

Dr. William B. Breed: This morning I read this case over once, and am therefore only one reading ahead of you. I shall set up the diagnosis that I have in mind, and perhaps between us we shall be able to support it — or discredit it. I will say, to begin with, that I think this man had portal cirrhosis of the liver with superimposed hematoma. Now let us read the material presented. Please give as much thought as you can to it with that diagnosis in view, and if you do not like the way things are going, please interrupt at any time.

* * *

Now that we have read the story again, I wonder if anyone is interested in the original diagnosis that I set up. (No answer.) Is anyone disinterested? (Silence.) This is an open forum; everyone's opinion has equal standing.

First of all, let us see if we can account for everything on the basis of syphilis, which has to be considered in view of the positive Hinton test. He obviously had liver disease. I do not know very much about diffuse syphilis of the liver. It is quite uncommon. Gummatous involvement of the liver is the commonest form of liver disease in syphilis, but such a manifestation should respond to iodides and bismuth, which apparently this did not. I see no other evidence anywhere of syphilis, such as aortitis, aneurysm or nervous-system involvement, and so I am willing to ignore the positive Hinton test as being the only indication of syphilitic infection and, therefore, not important as a cause of this man's illness and death.

Did he have metastatic cancer? That is the other most obvious alternative. As to cancer of the prostate, I noticed that the prostate was nodular but not tender. It does not say how hard it was. How long ago was this man in the hospital?

Dr. Tracy B. Mallory: About a year and a half ago.

Dr. Breed: Even a year ago it is probable that if they thought that the patient had cancer of the prostate with metastases they would have castrated him. Cancer of the prostate, moreover, does not as a rule metastasize to the liver. It may do so but the metastases are usually in the bones, in the lungs and in the brain. Is that not correct, Dr. Mallory?

Dr. Mallory: The bones above all.

Dr. Breed: Thank you. We have no evidence here to make a diagnosis of cancer of the prostate. He had urgency and frequency at one time, but we do not know whether it persisted. There

was no obstruction to the urinary output. I am not willing to make a diagnosis of cancer on the lone statement that he had a nodular prostate.

We have enough here to make a diagnosis of portal cirrhosis. He had ascites and a history of alcohol, even though his diet was said to have been adequate. It is too bad that we have no extremely low figure. Toward the end the patient accumulated fluid in his left pleural cavity. That is perfectly all right in the presence of cancer in the liver even though the fluid was not on the right side — the more usual site.

There are still some findings that I cannot adequately explain. The question is, Are most of

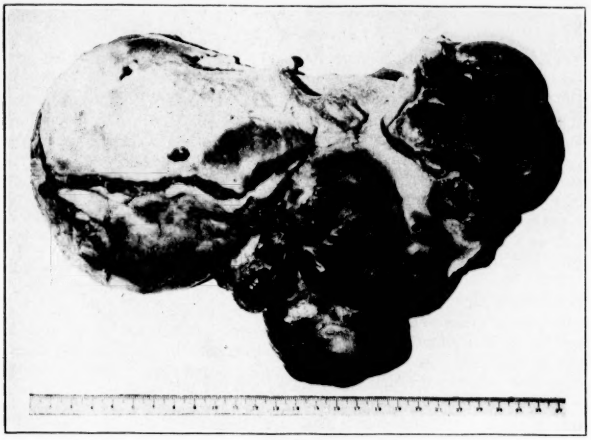

FIGURE 1.

x-ray films of the esophagus or some liver-function test. I am surprised that they were not done, because I suspect that he was discharged the first time with diagnoses of portal cirrhosis and syphilis. It seems to me that they should have tried a little harder to confirm the former.

What about the clubbing of the fingers? That is a hard one. I am going to say that it was congenital clubbing of the fingers. He had nothing on physical examination or by x-ray study to indicate a systemic cause of clubbing of the fingers, except possibly a congenital heart lesion. We know he had some enlargement and a loud systolic murmur on the left side of the sternum. He may have had a congenital lesion, but I doubt it. There were no other manifestations, such as cyanosis and typical murmurs. The reason I suspect he had cancer of the liver is the extreme degree of anemia and the rapid downhill course once he became sick. Apparently for five years he had had some fullness in the abdomen, which I explain on the basis of early portal cirrhosis, and during the last year of his disease he went downhill rapidly and developed a severe degree of anemia. The low white-cell count in the beginning is consistent with liver disease. The serum protein, if accurate, was depressed, but not to an

the things that appear here consistent with one diagnosis? Assume, for the moment, that we have ruled out syphilis and metastatic cancer from either the prostate or gastrointestinal tract, which by the way was not examined. That leaves us with malignant disease in the liver superimposed on what appears to be portal cirrhosis, and congenital clubbing of the fingers.

The peritoneoscopy does not help us at all, except to show that there had been adhesions and that the omentum had been pushed up against the liver to create a spontaneous omentopexy. I am going to assume that the mass in the left upper quadrant felt after paracentesis was an enlarged spleen. That is the commonest mass felt in the left upper quadrant. The hoarseness without obvious signs of paralysis does not interest me very much. There was no evidence of pressure on the trachea. There was only moderate edema of the arytenoid cartilages, and no paralysis of the cords. Aside from syphilis and metastatic cancer, what else may he have had? Has anyone here, having read this over with me, other suggestions as to the cause of this man's illness and death? (No answer.) Then I take it we all agree that I have mentioned at least three of the most likely diagnoses.

DR. RICHARD H. SWEET: Why is it necessary to assume that the patient had a primary cancer of the liver or hepatoma? Why does not cirrhosis account for the whole picture?

DR. BREED: I say that because he went downhill so rapidly, with such a severe degree of anemia and fluid in the chest.

DR. SWEET: That is inconsistent with cirrhosis alone?

DR. BREED: I believe so.

DR. ALLEN G. BRAILEY: The history covers a period of five years!

DR. BREED: We do not know how much anemia the patient had until seven months prior to death, when the anemia was discovered. How do you know he had anemia for a longer period of time? It says that for five years he had had swelling, that for two years there had been fullness and that for only a year previous to admission he had had these new symptoms. For a year and a half he was incapacitated, and in seven months he was dead.

DR. REED HARWOOD: I am not impressed with the rapidity of the patient's down-hill course. He had severe anemia — a red-cell count of 2,000,000 — seven months before he died. Seven months is a long period to live with an anemia of that severity due to cancer, is it not?

DR. MALLORY: There is one thing that is not clearly brought out in the summary. He had an enlarged spleen, but several examiners thought that they could feel another epigastric mass, which seemed to be attached in some way to the liver.

DR. BREED: We know by peritoneoscopy that he had a mass of omentum stuck up against the liver. Do you think one could have felt this mass of omentum through the abdominal wall, Dr. Sweet?

DR. SWEET: I doubt if one could unless there was something in it.

DR. BREED: Why, in all this time, if they felt an epigastric mass and suspected cancer or a lesion in the gastrointestinal tract, did they not investigate it? They could not have thought much of it because they sent him home with no diagnosis so far as the gastrointestinal tract was concerned. If they did that, how can I be expected to add to the diagnosis now? I also wonder a little bit why they did not think enough of the diagnosis of portal cirrhosis to go farther with it. Did they?

DR. BENJAMIN CASTLEMAN: Do you think that they may have made a diagnosis of syphilis on the first admission and sent him home on antisyphilitic treatment to see what would happen?

DR. BREED: That seems obvious. They sent the patient home under treatment with a diagnosis of syphilis. He did not do well and returned. Nothing further was done to establish a diagnosis. I still think that they should not have been quite so certain that this was syphilis, even though he had a positive Hinton test. I do not believe the diagnosis was accurate enough.

DR. MALLORY: Assuming that the history is correct and that he took never more than 3 or 4 ounces of whisky a day and usually less, do you still maintain that your diagnosis of portal cirrhosis is correct?

DR. BREED: I do not quite get the import of that question.

DR. MALLORY: I am asking a personal question, Dr. Breed. If the history of alcoholic intake is correct, do you want the patient to be proved to have portal cirrhosis?

DR. BREED: From a personal point of view? No. Seriously, however, I question the adequacy of his diet.

Have you any other suggestions that might set me right? I can see that the wind is blowing the wrong way. Is not someone here brave enough to come forward and help out Dr. Mallory? He has something on his mind. It is not because you do not want to speak up but because you do not know, is that right? (No answer.) Then I shall have to leave it that way.

CLINICAL DIAGNOSES

Portal cirrhosis.
Banti's syndrome.
Syphilitic cirrhosis?
Asymptomatic syphilis.

DR. BREED'S DIAGNOSES

Portal cirrhosis.
Hepatoma.
Latent syphilis (serologic).

ANATOMICAL DIAGNOSES

Syphilitic cirrhosis of liver (hepar lobatum).
Icterus.
Splenomegaly.
Ascites.
Hydrothorax, left.
Pulmonary atelectasis, left lower lobe.
Osteoarthropathy: fingers and toes.
Pulmonary tuberculosis, healed: apical, bilateral.

PATHOLOGICAL DISCUSSION

DR. MALLORY: Some years ago, I looked up all the cases of syphilis of the liver that had come to autopsy, and out of 20 cases, 18 were accidental findings at autopsy without referable symptoms in the clinical history. Two had symptoms; but in one of these there was amyloid disease, which may well have been responsible for the ascites, so that

left only 1 out of 20 patients who did have symptoms.

Syphilis of the liver is what this man had. The liver was considerably smaller than normal. It presented marked deep fissures dividing it into eight or ten lobes instead of the normal five (Fig. 1). You will note there is an almost pedunculated mass that dangles from the hypertrophied left lobe; this probably was at some time felt as the epigastric tumor. The shrunken right lobe is completely wrapped in omentum. When nodules are actually felt on a liver, it always means either tumor or syphilis of the liver. In a patient with symptoms of cirrhosis of five years' duration the recognition of palpable nodules on the lower margin of the liver would have led me to make the same diagnosis as Dr. Breed did; it seems the most reasonable one. I should hardly have expected a liver of this sort to have produced the characteristic symptoms of cirrhosis lasting over a period of five years.

I think the reason this patient made no improvement on syphilitic therapy is that the active stage of gumma formation was past history; when they started treating him he already had a scarred liver and one could hope to accomplish nothing with treatment.

There is no explanation of the marked hydrothorax on the left except the presence of ascitic fluid. Whenever there is ascitic fluid, it never surprises me to see a pleural effusion develop, although it is more apt to appear on the right than on the left; but it can develop on either side, as in the cases of ovarian fibroma that Dr. Joe V. Meigs has called to our attention.

We found no other evidence of syphilis. He had a normal cardiovascular system. He did not have anything wrong with the urinary tract.

DR. BREED: Was the clubbing of the fingers and toes congenital?

DR. MALLORY: I do not believe so. It was probably due to cirrhosis. A significant number of cases of cirrhosis of the liver have clubbing of the fingers.

A PHYSICIAN: What about the prostate?

DR. MALLORY: It was normal.

CASE 28442

PRESENTATION OF CASE

A forty-seven-year-old man was admitted because of severe abdominal pain.

The patient was known to have had a high blood pressure for many years but had been quite well until one month before entry. At this time he had an attack of moderately severe substernal pain, which lasted two days and then cleared completely. During the succeeding month he was well and active. Several hours prior to admission he developed an excruciating pain centered in the lower abdomen. At times he believed the pain radiated into the legs. There was no radiation of this discomfort to the chest. He vomited once. The patient collapsed soon after the onset of the pain and was immediately brought to the hospital. After reaching the hospital he noticed numbness of the right arm.

Physical examination revealed a semicomatose, thickset man lying quietly in bed complaining of severe lower abdominal pain. There was no cyanosis or dyspnea, and the neck veins were not distended. The examination of the lungs was negative. The apex impulse of the heart was felt in the fifth interspace, 11 cm. to the left of the midsternal line, which was 3 cm. beyond the midclavicular line. A rather harsh, moderately strong, systolic murmur was audible at the base of the heart in the aortic area and was transmitted to the neck, to the left of the sternum, and to the apex. A loud blowing diastolic murmur was heard in the aortic area, but was transmitted to the cardiac apex. The aortic and pulmonic second sounds were equal in intensity. The abdomen was soft and slightly protuberant, with rather generalized tenderness in the upper portion. A systolic murmur was prominent in the epigastrium; the peristaltic sounds were normal. The right arm was pulseless and slightly cooler than the left.

The blood pressure was 205 systolic, 95 diastolic, in the left arm; it could not be obtained in the right arm. The blood pressure was 220 systolic, 95 diastolic, in both legs. The pulse in the left arm was regular and of good quality, with a rate of 60. The temperature was 97.8°F.

An electrocardiogram showed a normal sinus rhythm with a rate of 50. T_1 was low and diphasic. The T waves were very low in Leads 2 and 4, and T_3 was high. P_3 was diphasic. There was a considerable amount of left-axis deviation.

Several hours after admission the patient said he felt better and indicated that the right arm was less numb and his feet less cold. The right hand and arm remained cool, but there was a weak but definite radial pulse. Four and a half hours later, less than twelve hours after reaching the hospital, the patient expired quietly within two or three minutes.

DIFFERENTIAL DIAGNOSIS

DR. REED HARWOOD: After reading the first two sentences I think we should consider the

possibility that this man had had a myocardial infarction. We might think then, if it were not for the numbness in his arm, that this episode of excruciating abdominal pain was the result of the separation of an embolus from a mural thrombus, which lodged in the bifurcation of the aorta. It is unusual for a man who has had severe pain to become semicomatose so suddenly. The fact that he had no cyanosis, dyspnea or signs in his lungs shows that he was not in congestive failure. The position of his heart suggests an enlarged left ventricle or possibly that the heart had been pushed to the left.

He might have had a systolic murmur for many years. He had had hypertension for a long time, and he might have had previous disease of the aortic valve. Probably he did not have the loud blowing diastolic murmur before the onset of his present illness. I say this because he was known to have had hypertension for a long time, and I should think that the doctor would have detected a loud blowing diastolic murmur. I think we can reason therefore that he did not have the diastolic murmur for more than a few hours. He probably did not have the systolic one for much longer either; otherwise, I think it would have been mentioned in the record.

Let us consider then the possible causes of a diastolic murmur in the aortic area. The first that comes to mind is syphilitic aortitis. He would have had a murmur of aortic regurgitation for a long time if he had had syphilitic aortitis. The same is true if he had had rheumatic heart disease with aortic regurgitation. I am going to exclude these two diagnoses on the basis that I believe that this murmur was of very short duration. Another possibility to be mentioned only to exclude it is rupture of the aortic cusp. I know very little about this condition, but I do not believe it would produce the numbness in the right arm and so forth. Although he had signs of aortic regurgitation, there is one very interesting finding — namely, the aortic and pulmonic second sounds were equal in intensity. I am going back to that and explain the probable mechanism when I have made my diagnosis.

The fact that the blood pressure in the legs was essentially the same as in the left arm rules out fairly well the possibility of a rider embolus.

I think we can interpret the electrocardiogram as follows: the interpreter was trying to find something abnormal, but he had to admit in the end that the patient did not have anything suggesting coronary thrombosis or myocardial infarction. It did suggest, however, that the patient had left ventricular hypertrophy, as shown by the axis deviation. This is consistent with the story of long-continued hypertension. The electrocardiogram, to my way of thinking, rules out quite well the possibility of myocardial infarction two months ago, and we can dismiss that diagnosis here and now.

In summary, the patient had severe pain in the abdomen, numbness and lack of pulse in the right arm and died suddenly. I can think of only one diagnosis that will fit the findings in this case and that is dissecting aneurysm. We might argue that the age, forty-seven, was rather young for that condition. However, I have seen cases reported considerably younger. So I do not believe we need to rule out the possibility of dissecting aneurysm on that single finding. Then there is the question of whether he had syphilis as a basis for his aneurysm. Dissecting aneurysm can occur in patients with syphilis in the aorta and cases have been reported. The late Dr. Soma Weiss, however, believed that if dissecting aneurysm did occur in a case of syphilitic aortitis it was coincidental rather than a result of the syphilitic process. In fact there is some suggestion that the inflammatory process of syphilis may protect the patient against this accident. I am going to say that he probably did not have it.

There is another interesting point that I want to discuss — that is, the matter of his being semicomatose. Patients with severe, sudden onset of pain in the abdomen and chest do not become comatose suddenly unless they have a sudden lowering of the blood pressure that is sufficient to produce cerebral anemia. We know that this patient did not have sudden lowering of the blood pressure, but may he not have had an anemia, at least of one cerebral hemisphere? We notice he was pulseless in the right arm. If he had a dissecting aneurysm, this means that the dissection must have involved the right innominate artery. In keeping with this is the fact that at the time when a weak but definite pulse could be found, he was not semicomatose and was able to say he felt better. Then, while he seemed to be improving, he suddenly died.

Dissecting aneurysms very frequently rupture somewhere along their course, and I think the most frequent location of rupture is into the mediastinum or into the pericardium. Either of these is rapidly fatal, and I do not see how we can distinguish between them. It is possible also that the aneurysm could have ruptured into the abdominal cavity, but in that case I do not believe he would have died so suddenly. So I am going to say that he had dissecting aneurysm of the aorta originating probably in the arch or in the descending portion of the arch, which dissected its way to the aortic ring and down through the abdomen and stopped somewhere in the region

of the bifurcation. He also probably had left ventricular hypertrophy.

I said that I was going to explain why he had the loud blowing diastolic murmur and still had an audible aortic second sound. Perhaps I should have said that I was going to try to explain it. I believe that the aneurysm distorted the aortic ring in such a way that a diastolic murmur was produced. Although the cusps clicked shut momentarily, it is probable that some blood did leak back into the heart.

Clinical Diagnosis

Dissecting aneurysm of aorta.

Dr. Harwood's Diagnosis

Dissecting aneurysm of aorta.

Anatomical Diagnoses

Dissecting aneurysm of aorta, with perforation into the pericardium.
Hemopericardium.
Media necrosis cystica aortica.
Nephrosclerosis.

Pathological Discussion

Dr. Mallory: The same diagnosis as Dr. Harwood's was reached on the ward and was substantiated at autopsy. There was an extensive dissecting aneurysm with a tear in the intima of the aorta about 3 cm. above the aortic valve. It dissected back as far as the annulus, partially surrounded the mouths of the coronary arteries but did not occlude them. It also dissected forward over the arch for a short distance into the innominate and left carotid arteries and 3 cm. down the left subclavian artery. In the subclavian, it had failed to rupture back through the intima so the subclavian was completely blocked by a projecting knuckle of intima. Dissection continued down the thoracic abdominal aorta into both iliac vessels, but in each of these rerupture of the intimal layer had occurred, so that the circulation was quickly re-established in both legs, if it was ever completely interrupted. There was a dissection for 1 cm. along the superior mesenteric artery, but again there was either no interference with the blood supply to the bowel or only a transitory one, because there was no discoloration of the intestines.

We found nothing to suggest that the aortic valve was not functioning perfectly well, and I have no clear explanation of the diastolic murmur. One is heard, however, every once in a while in these cases of dissecting aneurysm. It is at least possible that the jagged, rough tear in the intima creates a V-like flap between one column of blood in the old lumen of the aorta and the other column in the newly formed vessel outside. One can imagine it flapping back and forth to make an odd variety of murmurs, perhaps in both systole and diastole. The immediate mechanism of death, as Dr. Harwood prophesized, was rupture into the pericardium, with cardiac tamponade. The kidneys showed moderately severe nephrosclerosis.

Dr. Allen G. Brailey: What do you think of the coma? It was not shock?

Dr. Mallory: It is hard to see how it could have been shock when the systolic blood pressure was 220. I should think that the carotid artery may have been temporarily obstructed by external pressure. There was no extensive dissection along the carotid.

The aorta in this case showed a very good example of so-called "media necrosis cystica," which is often to be found in cases of dissecting aneurysm. It is a condition that occurs only in elastic arteries, and the dissection is ordinarily limited to arteries that have this elastic structure. For that reason, dissection extends only a very short distance along most of the branches of the aorta, such as the coronary and renal vessels, which are muscular arteries, but may extend several centimeters along the subclavian and iliac vessels, which have a structure similar to that of the aorta.

Dr. Brailey: Is this always associated with high blood pressure?

Dr. Mallory: I cannot answer that accurately.

Dr. Holmes: There was no evidence of syphilis?

Dr. Mallory: No; in a dissecting aneurysm, there almost never is.

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NAVAL MEDICAL OFFICER PROCUREMENT

Attention is called to a letter from the Office of Naval Officer Procurement, published elsewhere in this issue of the *Journal*, which emphasizes the need for obtaining medical officers from Massachusetts. Apparently, physicians who have previously been rejected because of minor physical defects may now receive favorable consideration.

MASSACHUSETTS LABORATORY TECHNOLOGISTS RESERVE CORPS

A LETTER in this issue of the *Journal* calls attention to an extremely necessary addition to the activities of the Massachusetts Committee on Public Safety. Because of an acute shortage of adequately trained laboratory technologists, many hospitals in the Commonwealth lack the specialized services that would be required in the event of wholesale bombing or other civil disasters. To correct this situation, the Committee on Public Safety is organizing the Massachusetts Laboratory Technologists Reserve Corps. By this means, each of the one hundred and twenty-four emergency (casualty receiving) hospitals in Massachusetts will be assigned technicians up to its needs, sometimes as many as three or four, for specialized work in an emergency.

The corps, which will consist of women trained as hospital technologists, will be organized in a manner similar to that of a military group. The members are expected to number approximately three hundred and fifty. The personnel will include both experienced technologists not at present engaged in such work and those who are already employed. Under the direction of nine regional directors, the corps will function in all parts of the Commonwealth.

The Massachusetts Committee on Public Safety is to be congratulated for its zeal in preparing the Commonwealth against possible disaster. The formation of this corps is a vital contribution to civilian defense.

THE SENESCENT SKIN

Now that the older age groups constitute a large proportion of the population, more interest has been aroused in geriatrics, but as yet, few phases of this branch of medicine have been explored to any extent. Even the most accessible organ — the skin — has been given little study.

In this issue of the *Journal*, attention is called to the many varied conditions that contribute to generalized itching in old persons. Not only local causes but also general factors, both external and internal, act to produce this symptom, which can have a very serious effect on the patient: general pruritus may be an early manifestation of a serious disease, when, by interfering greatly with sleep

and nutrition, it may thus act to lower further the resistance of a patient already combating the inroads of a disease of other organs. In general, eczema, keratoses of various types, pruritus and cancer are the commonest diseases of the skin of old persons. At the same time, it should not be forgotten that other skin diseases — those of childhood and of middle life — may be found in people of this age group and that the diagnosis may be missed by reason of failure to consider such possibilities.

Grossly, as the years pass, the skin becomes dry, thinner, less elastic and wrinkled. Pigmented areas, dilated vessels and small scaly areas may develop. Histologically, according to Hill and Montgomery,* the greatest changes occur in the collagen and elastic tissue, with some atrophy of the rete pegs. Investigation concerning the physiologic and chemical changes in the skin of this older group has received but slight attention. In addition, there is little knowledge of the effect on the skin of senile circulatory changes — the failing heart muscle, the fibrotic changes in arteries and the loss of elasticity of the veins and capillaries. Such changes cannot fail to have some relation to morbid processes in the skin. Whether the reduction in glandular activity and the subsequent dryness are results of circulatory change is not known. The effect of the aging process elsewhere or of pathologic processes in other organs is obscure. Much remains to be learned of the part played by the nervous system, by hormones and vitamins and by nutrition in aging of the skin. Truly, a large realm of investigation lies open to the explorer in this field.

*Hill, W. R., and Montgomery, H. Regional changes and changes caused by age in normal skin: histologic study. *J. Invest. Dermat.* 3:231-245, 1940.

MEDICAL EPONYM

Scanzoni Maneuver

The description of this maneuver appears in "Die geburtschilflichen Operationen [Obstetric Operations]," which was issued by its author, Professor Friedrich Wilhelm von Lichtenfels Scanzoni (1821–1891), of Würzburg, in the form of a reprint, with revisions, of a portion of the third volume of his *Lehrbuch der Geburtshilfe* [*Textbook of Obstetrics*] (1852). A portion of the translation follows:

> Our procedure under these circumstances is as follows: If the head lies with the brow anterior and turned to the left, so that the longitudinal suture runs in the right diagonal diameter, the left blade is applied in front of the left sacroiliac synchondrosis, the right behind the right oval window [*eirunder Loche*]. . . . The head is then rotated, by turning the instrument from right to left through one eighth of a circle, so that the longitudinal suture lies parallel with the transverse diameter of the pelvis.
>
> Both blades of the forceps are now removed and reapplied so that the left blade lies behind the left oval window, the right in front of the right sacroiliac joint, whereby the occiput may be drawn under the pubic arch by again rotating the instrument.

R. W. B.

MASSACHUSETTS MEDICAL SOCIETY

COMMITTEE ON MATERNAL WELFARE

Analysis of Causes of Maternal Death In Massachusetts during 1941

Heart Disease

In the tabulation that appeared in the first installment of the maternal-mortality study for 1941, the deaths of 42 women were attributed to medical causes. These can be broken down into the following groups:

Heart disease	22
Pneumonia	12
Hypertensive nephritis	2
Meningitis	2
Gastric ulcer	2
Tuberculosis	1
Colitis	1
Total	42

Of the 22 deaths that occurred as a result of heart disease, in 8 the patient was undelivered, 8 followed normal delivery, 5 followed operative delivery and 1 was associated with miscarriage. Autopsy was performed in only 1 case.

The undelivered cases emphasize the seriousness of heart disease during pregnancy and prove beyond question that heart disease during pregnancy alone may cause death. From this standpoint, the deaths are, of course, preventable, and the medical profession is responsible for them if its advice was sought and abortion not advised. If these patients had been told to avoid pregnancy and if they had followed this advice, the deaths would not have occurred. Religious beliefs, however, on the part of the physician or the patient may well have played a part in these fatalities.

The cases of the 8 patients who died undelivered are reviewed briefly.

The first was a patient who, at three months, developed symptoms of heart failure; she was admitted to the hospital two months later with cardiac decompensation and died undelivered, in spite of treatment. Although no autopsy was performed, this patient was known to have had rheumatic heart disease and should have been warned against becoming pregnant; and, in the event that pregnancy did take place, abortion should have been performed in the early stage.

The second case was that of a patient who had had two previous pregnancies and who was known to have a definite heart lesion; in her third pregnancy she had attacks of tachycardia and palpitation. When seven months pregnant she became acutely decompensated, entered the hospital and died a few hours later. The criticism of the first case can be reiterated here.

The third patient, who had a history of a cardiac lesion since childhood, was a primipara; she was being prepared for a cesarean section when she had acute cardiac failure and died fifteen minutes later. There is little to be said in criticism of this case.

The fourth patient was known to have had rheumatic heart disease with a double mitral lesion for years. She was seen at first by her physician when about five months pregnant, at which time she was in cardiac distress, but refused hospitalization. When seen a week later at home, she was in extremis and died shortly. This fatality can be attributed either to the patient's ignorance in not having sought medical advice earlier in pregnancy or to religious dogma. She should never have become pregnant.

The fifth case in this group was a patient who, with a history of rheumatic heart disease since childhood, was seen first at three months and again at six months, when, because of signs of decompensation, she was sent to the hospital. Her stay there lasted only six days. She should not, of course, have been allowed to go home, but she was said to have been unco-operative. When she was approximately at term, acute cardiac decompensation developed and she died undelivered. The treatment of this case was obviously unintelligent.

The sixth patient, a multipara with rheumatic heart disease, did not consult a physician until near term, at which time she had an attack of acute cardiac failure and was practically moribund at the time of his arrival. This patient was not in labor and died undelivered en route to the hospital.

In the two other cases in this group, the records are not adequate. One patient is known to have had rheumatic heart disease of long standing; she died undelivered at six months. The other had the same history of rheumatic heart disease and died near term.

The following are the records of the 8 patients who died of heart disease following normal delivery. The first was a patient who, at six months, was hospitalized and given digitalis for cardiac decompensation and who was sent home six weeks later. She re-entered the hospital at approximately eight months, being in poor condition and in mild labor. A living 4½-pound baby was delivered, but the mother went steadily downhill and died on the fifth post-partum day.

The second patient was delivered normally but developed cardiac insufficiency seven weeks post partum; in spite of treatment, she died five days after the onset of the attack.

The third patient, a multipara who gave a history of rheumatic heart disease, entered the hospital at about eight months with cardiac decompensation associated with psychosis. She delivered herself spontaneously at term; but marked decompensation immediately followed delivery, and she lived only forty-eight hours.

The fourth patient delivered herself at term after a very short labor. Since she had rheumatic heart disease, she had been warned against pregnancy; she was hospitalized at seven months because of decompensation; she improved and remained in the hospital until she went into labor at term. Following a normal delivery, she again became decompensated and died two weeks later.

The fifth case was that of a primipara with a history of rheumatic heart disease who developed definite signs of bacterial endocarditis after a short, normal labor, from which death resulted four weeks later.

The sixth patient was a multipara who had known cardiac disease associated, during her last pregnancy, with an elevated blood pressure. Labor was induced at term by rupturing the membranes, a living 9½-pound child being delivered seven hours later. Death occurred four hours after this, following the appearance of pulmonary edema.

The last two cases, whose histories are not complete, concerned patients who had long-standing rheumatic heart disease; one developed subacute bacterial endocarditis after a spontaneous delivery, and the other, having received no medical attention whatsoever during her pregnancy, succumbed from cardiac failure several hours after delivery.

Of the 5 cases of operative delivery, the first patient was delivered prematurely at seven months. At six months she developed subacute bacterial endocarditis, proved by blood culture to be due to *Streptococcus viridans*, for which she was given sulfathiazole. She started in labor at seven months and was delivered by low forceps. Death occurred a month later. There is little to be said in criticism of the handling of this case. The patient's history shows that she had had chronic endocarditis, upon which the subacute bacterial endocarditis developed.

The second patient, delivered by low forceps, was one who had rheumatic heart disease of long standing and developed arterial emboli, proved by autopsy, on the ninth post-partum day. She died thirty-four days after delivery.

The third case in this group was a patient with congenital heart disease on whom a planned cesarean section was done. Although this patient had adequate prenatal care, she did not survive the operation (which was done under local anesthesia), death occurring three days later. At the beginning of this pregnancy, the question of whether or not she should be aborted was raised, but after consultation it was decided that the cardiac condition was compatible with a safe pregnancy.

The fourth patient had rheumatic heart disease and died shortly after delivery of cardiac decompensation; and the fifth patient, whose history is inadequate, died in heart block during a low-forceps delivery.

The miscarriage in this series occurred spontaneously at two months in a patient who had rheumatic heart disease; death due to congestive heart failure occurred two weeks later. Nature did the best she could for this patient.

These 22 fatal cases of known heart disease complicating pregnancy represent a little more than 9 per cent of the total maternal deaths occurring during 1941. They show how serious heart disease is in pregnancy. They emphasize the importance of adequate prenatal care, which many of these patients did not have. They bring up the questions of abortion, of sterilization and of intelligent contraceptive advice. All these problems must be met if the maternal mortality from this particular complication is to be lowered in Massachusetts.

ESSEX NORTH DISTRICT MEDICAL SOCIETY

Report of Special Committee on Malpractice Insurance

Investigation of malpractice insurance in Essex County was prompted mainly because of the statement by one insurance-company official that Essex County was a hotbed for malpractice suits. Two other reasons that led to this study were as follows: first, refusal of one company — the Medical Protective Company, Fort Wayne, Indiana — to write for amounts greater than $5000–$15,000 limits, although this same company admitted writing higher limits outside of Essex County; and, second, the matter of premium rates — considerably higher than in former years and not the same in all companies.

We wrote to the medical societies of several nearby states to ascertain their experience with this type of insurance.

The Connecticut State Medical Society has a group-plan policy with the Aetna Life Insurance Company. Their experience with this group plan has been highly satisfactory. The limits are $10,000–$25,000. The rate for minimum coverage — no surgery — is $12.50. The rate for unrestricted practice is $16.50.

The Medical Society of the State of New York also has a group plan, with the Yorkshire Indemnity Company. Fifty to sixty per cent of the members of the society are insured under this plan. The rate is $28.00 for $5000–$15,000 limits, and $36.40 for $10,000–$30,000 limits. These rates include all types of practice except x-ray for therapeutic purposes. The Yorkshire Indemnity Company has an agreement with the society whereby a committee from the society may inspect all the books and records of the company whenever desired. As a result of three years' experience, it was found possible to reduce the base rate $2.00. The premium rate for x-ray therapy was dropped to $58.00 from $70.00 — quite a difference. Complete understanding and co-operation between the society and the Yorkshire Indemnity Company make immediate rate reduction possible whenever the figures warrant it.

The Rhode Island Medical Society has a group policy with the United States Fidelity and Guaranty Company, paying $20.00 for $5000–$15,000 limits; practice in medicine and surgery is unlimited.

In October, 1941, to get a cross section of the amount of malpractice business each company was writing in one city, we made a survey of Haverhill, and found as follows: American Motorists Company, 7 policies in force; Medical Protective Company, 12 policies; and United States Fidelity and Guaranty Company, 22 policies. Four physicians were found to carry no malpractice insurance.

The Aetna Insurance Company retired from the malpractice field in Massachusetts in 1920, because of poor experience.

The Medical Protective Company, which has probably had the largest share of this type of business in Essex County for the last several years, found that in one recent year more than 30 per cent of the claims pending in Massachusetts were against physicians in Essex County. The last check made by them showed a reduction of this figure to 20 per cent. They give no satisfactory explanation of this preponderance of claims in Essex County except to say that the people of this county are more suit-minded.

As to reliability, record and other pertinent factors, the four companies mentioned above line up somewhat as follows in the opinion of the committee (the comparative rates are listed in Table 1):

> The *Medical Protective Company* is relatively small as regards its capital (assets, $2,883,700). However, they are specialists in the medical defense field, doing no other kind of business. In Boston they have an attorney — Mr. Charles Dunn, who has had much ex-

perience in medical defense. Dr. Arthur W. Allen, chairman of the Committee on Medical Defense of the Massachusetts Medical Society, states that the record of this company in this state is good. As has been mentioned, their suit experience in Essex County is very high.

The *United States Fidelity and Guaranty Company* (assets, $30,000,000) has not had a high suit rate in Essex County. They retain the law firm of Powers and Hall, in Boston, who have had a wide experience in defending malpractice suits. In their policy they have a dissolve attachment bond feature. Dr. Allen says that their record in this work is good. This company advises minimum limits of $10,000–$30,000, and believes so strongly that these limits are necessary that it does not write for less. On the other hand, the Medical Protective Company believes that $5000–$15,000 limits are sufficient, and will not write for more in Essex County.

The *American Motorists Company* (assets, $10,000,000), a subsidiary of the Lumbermens Mutual Insurance Company (assets, $41,000,000), is relatively new in the medical-defense field, although they have been in Massachusetts for ten years and now collect $13,000 in premiums for this type of insurance. They advise $10,000–$30,000 limits, but will write for less.

The *Metropolitan Casualty Company* (assets, $11,000,000) has been official for the Massachusetts Dental Society for nearly twenty-five years. A prominent feature of their policy provides payment of premium on appeal bonds without limit. This company pays $15 a day for not more than seven days to the defendant doctor for loss of time while in court.

While this study was in progress, and after one set of rates had been offered, Ladd and Milne, in Haverhill, obtained the following concession from the American Motorists Insurance Company, which they represent: for all physicians in Essex County, whether engaged in general practice, major surgery or another specialty, but excluding those physicians using x-ray and radium for therapeutic purposes, the premium of $20.00 for $5000–$15,000 limits is offered; for $10,000–$15,000 limits, $24.00; and for $10,000–$30,000 limits, $26.00. The company states that if a substantial amount of the business in this county is obtained over a period of one year, the rate will continue. No assessment can be made against the policyholder, and a dividend of 20 per cent, if earned, is returned to him. It is a legal reserve stock company, but is owned and operated by the Lumbermens Mutual Insurance Company. According to Ladd and Milne, the company offers this rate schedule only through their agency.

In summary, this committee chooses to recommend no one company for all the members. It is hoped that from the facts presented, each member may better select that insurance company which meets his individual needs best. Reliability is paramount, experience in the field is of considerable importance, and a reasonable rate is desired.

The committee will gladly assist any member by allowing him to examine any of the data and all the letters that we have collected as a result of this investigation.

GUY L. RICHARDSON, M.D., *Chairman*
GEORGE J. CONNOR, M.D.
ALDEN B. GEORGE, M.D.

TABLE 1. *Comparative Rates for Malpractice Insurance.*

COMPANY	GENERAL PRACTICE (NO SURGERY)		GENERAL PRACTICE SURGERY AND DIAG. X-RAY		GENERAL PRACTICE AND THERAP. X-RAY	SPECIALISTS IN X-RAY	SPECIALISTS IN SURGERY, OBSTETRICS AND ORTHOPEDICS	SPECIALISTS IN E., E., N. AND T.
	$5–10,000	$10–30,000	$5–10,000	$10–30,000	$10–30,000	$10–30,000	$10–30,000	$10–30,000
Medical Protective Company	$24.00	—	$32.00	—	—	—	—	—
United States Fidelity and Guaranty Company	—	$20.00	—	$40.00	$60.00	$70.00	$60.00	$40.00
American Motorists Company	$20.00*	$26.00*	$20.00*	$26.00*	—	—	—	—
Metropolitan Casualty Company	—	$20.00	—	$40.00	$60.00	$70.00	$60.00	$40.00

*Less 20 per cent, if earned.

NEW HAMPSHIRE MEDICAL SOCIETY

DEATHS

COTTON — CURTIS B. COTTON, M.D., of Wolfeboro, died August 6. He was in his seventy-eighth year.

Dr. Cotton, the son of Joel F. and Elivna (Gilman) Cotton, was born in Moultonboro and graduated from the University of Vermont College of Medicine in 1888. He was a member of the New Hampshire Medical Society and the American Medical Association.

He is survived by his widow, a son and a sister.

ROWE — ARTHUR J. ROWE, M.D., of Penacook, died September 9. He was in his seventy-fifth year.

Born in South Barnstead, the son of Dr. James W. and Emma (Clark) Rowe, Dr. Rowe received his degree from Dartmouth Medical School in 1899. He was a member of the New Hampshire Medical Society and the American Medical Association.

He is survived by his widow, Mrs. Ida (Avery) Rowe, three brothers and a sister.

WAR ACTIVITIES

PROCUREMENT AND ASSIGNMENT SERVICE

The following release was recently received from the Washington office of the Procurement and Assignment Service:

> The Directing Board of the Procurement and Assignment Service is pleased to announce that 95 per cent of the 1942 procurement objective of medical officers for the armed forces has already been met. Toward this total a number of states have supplied more than their share of physicians. Only a few states are lagging behind in their quotas, and it is from these states that the additional physicians needed during the current year should come.
>
> The recruitment of such a large number of physicians in a few months is a remarkable achievement and another demonstration of the traditional patriotism and unselfishness of the medical profession. In this achieve-

ment, and particularly in those of its members who are "in service," the profession can justifiably take pride.

The end, of course, is not yet. Increases in the armed forces will necessitate more medical officers, and additional demands will be made upon the profession for medical services in critical war-production areas. The Directing Board is convinced, however, that the physicians of this country will respond to future calls for service, whatever they may be, in the same splendid manner with which they have already volunteered for service with the armed forces.

FRANK H. LAHEY, M.D.
HAROLD S. DIEHL, M.D.
HARVEY B. STONE, M.D.
JAMES E. PAULLIN, M.D.
C. WILLARD CAMALIER, D.D.S.

MISCELLANY

NATIONAL AWARD TO MASSACHUSETTS HOSPITALS

The American Hospital Association, at its recent meeting in St. Louis, awarded to the Massachusetts Hospital Association and to the Blue Cross a silver cup in recognition of the best state-wide observance of National Hospital Day on May 12. Dr. Charles F. Wilinsky, president of the Massachusetts Hospital Association, accepted the cup in behalf of the hospitals that participated in the observance.

On their return to Boston, Dr. Wilinsky and Mr. R. F. Cahalane, executive director of the Blue Cross, whose organization directed the program, were received by Governor Saltonstall, who stated: "It is a great honor for the hospitals of Massachusetts to be singled out among those of the entire country for this national award. It only serves further to confirm my belief that the hospitals of the Commonwealth will continue to administer in wartime, as in peacetime, to the needs of the civilian population."

The cup will remain the permanent property of the two organizations, and is on view at the Blue Cross headquarters, 230 Congress Street.

MASSACHUSETTS HOSPITALS GET NATIONAL AWARD

Governor Saltonstall views the cup that was awarded by the American Hospital Association to the Massachusetts Hospital Association and the Blue Cross for the best statewide observance of National Hospital Day. Holding the cup with the Governor is Mr. R. F. Cahalane and in the center is Dr. Wilinsky.

CORRESPONDENCE

NAVAL MEDICAL OFFICER PROCUREMENT

To the Editor: A recent letter received from the Navy Department directs the Office of Naval Officer Procurement not to accept applications for commissions in the Medical Corps, United States Naval Reserve, from physicians of the New England States other than Massachusetts,

as the other states have met their quotas set by the Procurement and Assignment Service, War Manpower Board.

This directive also informs us that we should procure as many physicians from Massachusetts as possible, rejecting only those who have not been cleared by the Procurement and Assignment Service, those not meeting the professional requirements, and those with physical defects that, in the opinion of the Senior Medical Officer, would constitute a liability to the Government.

Those physicians from states other than Massachusetts who have *already been cleared* as available for military service will have their applications forwarded.

LIEUTENANT COMMANDER ROBERT S. PALMER, (MC) USNR
Senior Medical Officer
150 Causeway Street
Boston

MASSACHUSETTS LABORATORY TECHNOLOGISTS RESERVE CORPS

To the Editor: At the suggestion of medical and hospital authorities of the Massachusetts Committee on Public Safety, the Massachusetts Technologists Reserve Corps — consisting of approximately three hundred and fifty women trained as hospital technologists — is being organized under the direction of a special committee. The one hundred and twenty-four emergency (casualty receiving) hospitals in Massachusetts will be assigned technicians up to the needs of each hospital, sometimes as many as three or four, for specialized work in the event of civil disaster; at present, there is a serious shortage of adequately trained laboratory technologists.

The organization of the corps will be somewhat like that of a military group, with state, regional and local heads. A chief medical technologist will be assisted by subordinate technologists, and technicians' aides will be enrolled in the near future. All members will wear standard insignia on their regulation laboratory uniforms.

The corps will be established by a special committee appointed by Dr. A. William Reggio, Medical Director of the Massachusetts Committee on Public Safety. Nine regional directors will be in charge of enrollment in their respective areas.

The personnel of the corps will be recruited from women who have had experience or are currently employed in hospitals, public-health laboratories, technical work for private physicians, industrial hygiene or pharmaceutical houses. They will be urged to spend two or three hours a week in refresher courses, unless they can demonstrate that they are already fully qualified for such work.

The regional directors, to whom inquiries concerning the corps should be addressed, are as follows:

Region 1. Dr. Helen M. Scoville, 741 North Street, Pittsfield
Region 2. Dr. Fred D. Jones, 20 Maple Street, Springfield
Region 3. Dr. James S. P. Beck, 119 Belmont Street, Worcester
Region 4. Dr. D. A. Nickerson, Salem Hospital, Salem
Region 5. Dr. Charles F. Branch, 750 Harrison Avenue, Boston
Region 6. Dr. Helmuth Ulrich, 99 Bay State Road, Boston
Region 7. Miss E. K. Jones, Supt., Cape Cod Hospital, Hyannis
Region 8. Dr. Isabel M. Wason, 101 Page Street, New Bedford
Region 9. Dr. Edwin St. John Ward, Templeton

The co-operation of all physicians in the Commonwealth is requested to make this program effectual, and it is hoped that physicians who know of potential laboratory technicians will refer them to the proper regional directors.

DONALD A. NICKERSON, M.D., *Chairman*
Laboratory Technician's Committee
Salem Hospital
Salem, Massachusetts

NOTICES

JOSEPH H. PRATT
DIAGNOSTIC HOSPITAL
Bennet Street, Boston
Lecture Hall, 9–10 a.m.

MEDICAL CONFERENCE PROGRAM, NOVEMBER

Tuesday, November 3 — Some Problems in Malaria. Dr. Edward I. Salisbury.
Wednesday, November 4 — Dermatological clinic. Dr. Francis M. Thurmon.
Friday, November 6 — Changes in Chloride Metabolism. Dr. George W. Thorn.
Friday, November 13 — Selected Problems in Aviation Medicine. Dr. Ross A. McFarland.
Wednesday, November 18. Peptic Ulcer. Colored motion picture — courtesy of John Wyeth and Brother, Incorporated.
Friday, November 20 — Clinicopathological conference. Dr. Chester S. Keefer and Dr. H. E. MacMahon.
Wednesday, November 25 — Treatment of Tuberculosis. Dr. Joseph H. Pratt.

On Tuesday and Thursday mornings (except November 3) Dr. S. J. Thannhauser will give medical clinics on hospital cases.

MASSACHUSETTS PSYCHIATRIC SOCIETY

The annual meeting of the Massachusetts Psychiatric Society will be held at the Hotel Kenmore, Boston, on Monday, November 9, at 6:30 p.m. The guest speaker will be Dr. Edward A. Strecker, professor of psychiatry, University of Pennsylvania, whose topic will be "Treatment of Neuropsychiatric Disabilities."

GREATER BOSTON MEDICAL SOCIETY

A meeting of the Greater Boston Medical Society will be held in the auditorium of the Beth Israel Hospital on Tuesday, November 3, at 8:15 p.m.

PROGRAM

Present Status of Sex-Hormone Therapy. Dr. Joseph C. Aub. Discussion by Drs. H. B. Friedgood, Samuel Gargill and Henry Finkel will follow.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY

The next written examination and review of case histories (Part I) for all candidates will be held in various cities of the United States and Canada on Saturday, February 13, 1943, at 2:00 p.m.

Arrangements will be made so far as possible for candidates in military service to take the Part I examination (written paper and submission of case records) at their places of duty, the written examination to be proctored by the commanding officer (medical) or some responsible person designated by him. Material for the written examination will be sent to the proctor several weeks in advance of the examination date. Case records may be submitted in advance of the above date only by candidates in